VOLUME 46 NUMBER 3, PART I SEPTEMBER, 1958

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THIRD SERIES FOUNDED BY EDWARD JACKSON

CONTENTS

DEPARTMENTS

For a complete table of contents see page xxv

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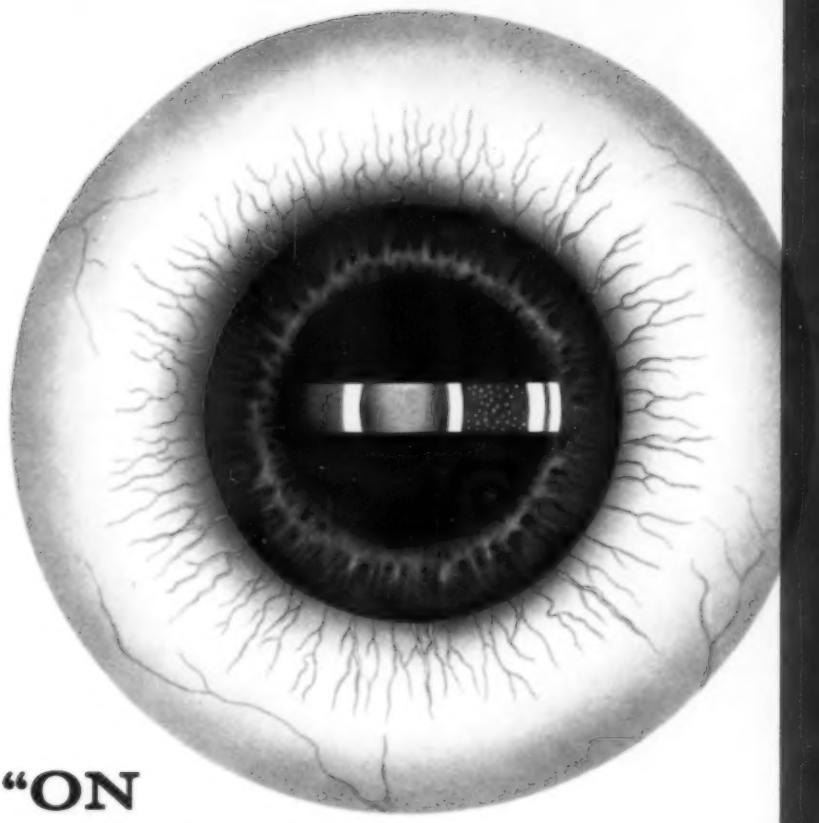

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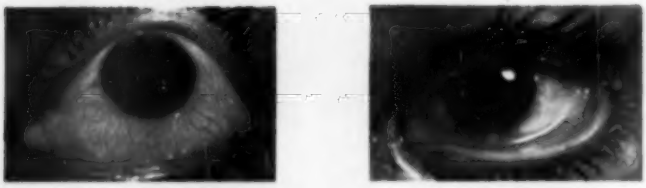

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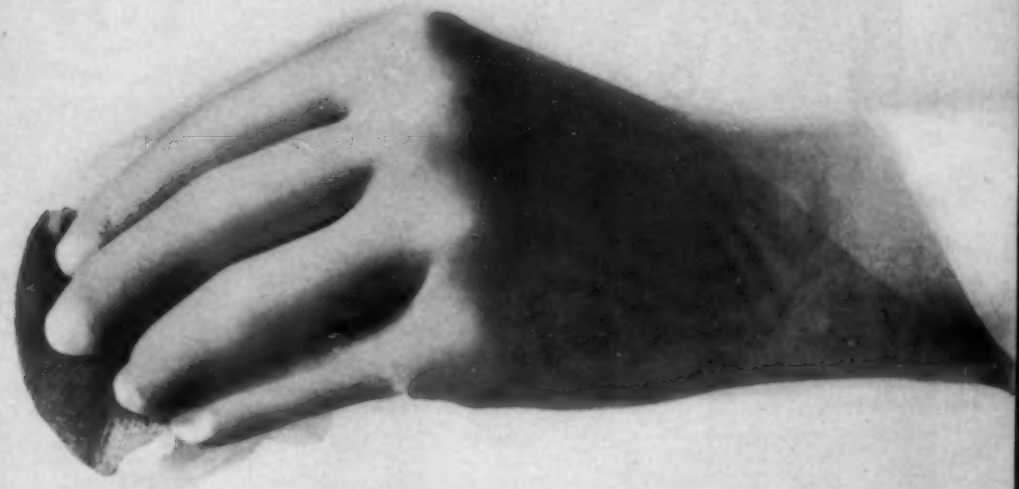

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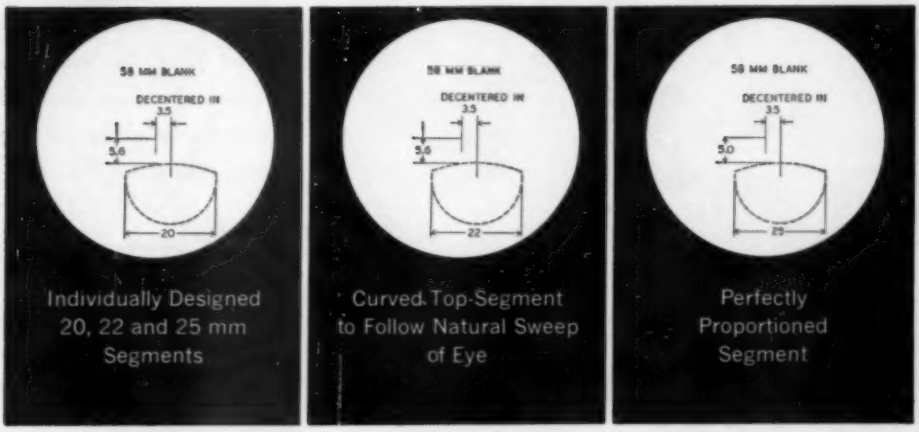

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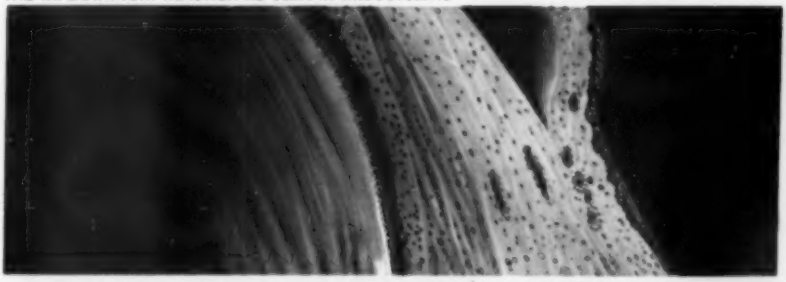
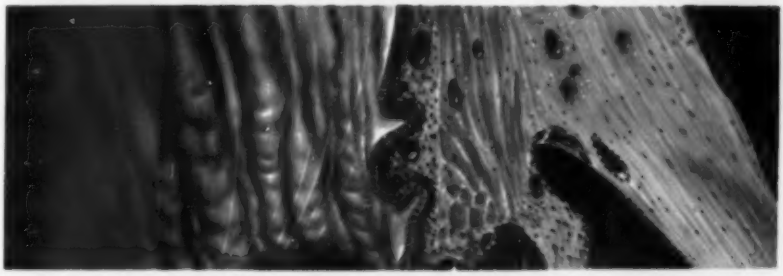
EXUDATIVE INFLAMMATION OF THE UVEAL TRACT

THE INFLAMMATORY REACTION AS SEEN IN IRIDOCYCLITIS

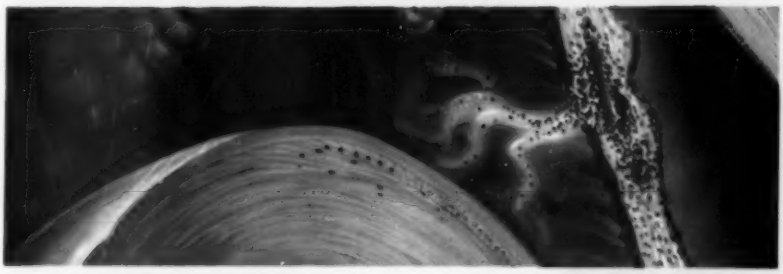
A. Normal tissue. From left to right: posterior surface of ciliary ring, ciliary retina, pigmented layer, ciliary muscle, sclera, and conjunctiva.



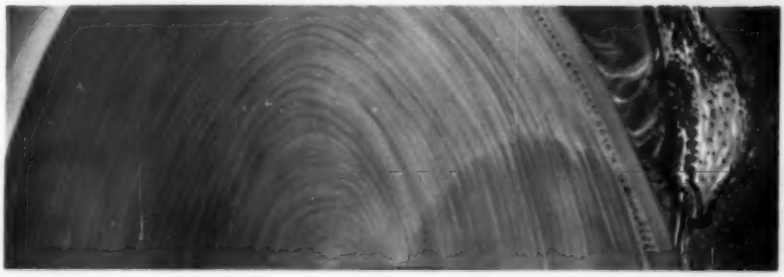
B. Hyperemia is the result of reaction to an endogenous toxin or allergen. Blood vessels are dilated. There is heightened local heat and color. Escape of blood plasma causes edema.



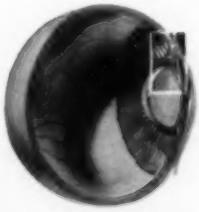
C. The next stage is that of exudation, shown here as macrophages and fibrin streaming from the posterior surface of the iris.



D. As the condition becomes subacute, there is concomitant active exudation and repair. With chronicity, fibroblasts proliferate, new capillaries develop, and various kinds of white cells appear. Formation of a posterior synechia is shown.



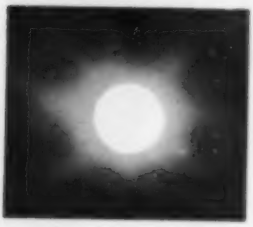
SCHWENK



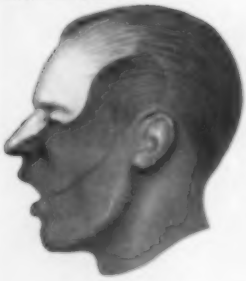
A SYNOPSIS OF OCULAR INFLAMMATORY DISEASE (IRIDOCYCLITIS)

Subjective symptoms:

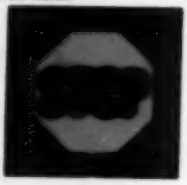
Photophobia



Trigeminal pain



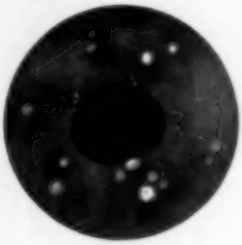
Blurred vision



Lacrimation

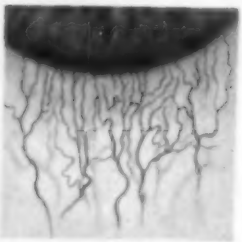
Clinical findings:

Hazy iris



Swollen upper lid

Ciliary injection



Sequelae: (if untreated)

Anterior and posterior synechiae

Lenticular changes

Secondary glaucoma

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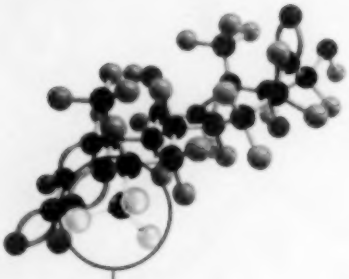

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Medrol's chemical distinction is reflected in
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- fewer and milder side effects than with any other corticoid

1. Feinberg, S. E.: Medrol in Allergic Conditions: Clinical and Experimental Findings, Metabolism 7:477 (July) 1958.
2. Gordon, D. M.: Methylprednisolone in Ophthalmology, Metabolism 7:569 (July) 1958.

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'DARANIDE'
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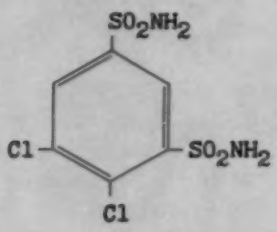
Dichlorphenamide

DESCRIPTION

'Daranide' is a new, orally effective, potent carbonic anhydrase inhibitor. Because of its marked inhibitory effect on this enzyme in the eye, 'Daranide' is a therapeutic agent of considerable value in reducing the increased intraocular pressure in many types of glaucoma.

CHEMISTRY

'Daranide', a dichlorinated benzenedisulfonamide, is known chemically as 1,3-disulfamyl-4,5-dichlorobenzene and is designated by the generic name of dichlorphenamide. It has the following structure:



MODE OF ACTION

'Daranide' is primarily a carbonic anhydrase inhibitor; i.e., it interferes with the catalytic activity of this enzyme, which is concerned with the formation of H^+ and HCO_3^- ions and the conservation of base as sodium bicarbonate. Under the influence of 'Daranide' the renal excretion of sodium, potassium, and bicarbonate is increased and an alkaline urine is produced. However, this compound also has the

advantage of causing a definite increase in chloride excretion. Partially as a result of the greater excretion of chloride, 'Daranide' does not produce metabolic acidosis as readily as other compounds of this nature. Furthermore, in animal studies 'Daranide' has been shown to maintain its essential activity over the course of weeks or months.

The compound does not appear to be concentrated in gastric mucosa in sufficient amounts to influence materially the activity of carbonic anhydrase in this tissue. Thus, in dogs, gastric motility or secretion is not affected to any significant degree by the administration of 'Daranide'.

PHARMACOLOGY

'Daranide' is uniformly and well absorbed following its oral administration; it is essentially as active when given orally as parenterally. The enhancement of sodium, potassium, and chloride excretion by the oral administration of a single dose is rapidly manifested and persists for a number of hours, indicating a prolonged duration of action of the compound. The urinary pH is elevated as the result of sustained bicarbonate excretion. However, greater chloruresis and less acidosis are induced than with other carbonic anhydrase inhibitors, with consequent greater efficacy on repeated administration. The natriuretic effect of 'Daranide' has been observed even after 10 weeks of continued administration to dogs. The activity of 'Daranide' is not impaired in the presence of an acid urine such as that accompanying the administration of ammonium chloride.

'Daranide' has a low inherent toxicity when administered orally or parenterally to laboratory animals. The compound has been given orally to dogs and rats over a period of at

least six months with essentially no evidence of toxicity. Pharmacologic investigation indicates an absence of irritating effect on the mucosa of the eye as demonstrated by a lack of local anesthetic activity or irritation when a 1 per cent concentration of the drug (suspended in tragacanth) is instilled into the conjunctival sac of the rabbit eye.

CLINICAL EVALUATION

Effect on Intraocular Tension

Studies in humans have demonstrated that 'Daranide' administered orally reduces intraocular pressure in both glaucomatous and nonglaucomatous eyes. The onset of action following a single dose is within an hour and maximal effect is observed within two to four hours. The lowered intraocular tension is maintained for approximately six hours. It has been possible to control excessive intraocular pressure in patients with glaucoma for a period of many months by the daily combined use of 'Daranide' and miotics. Tonographic studies have shown that 'Daranide' lowers intraocular pressure by inhibiting the formation of aqueous humor in normal as well as in glaucomatous eyes.

Effect on Electrolytes

In the human as in the laboratory animal, 'Daranide' enhances the excretion of sodium, potassium, bicarbonate, and water. An increased chloride excretion greater than that following the use of other carbonic anhydrase inhibitors is also observed with 'Daranide'. This enhanced excretion of chloride explains, at least in part, the continued efficacy of 'Daranide' with repeated administration and the lesser propensity for the development of metabolic acidosis.

As is the case with all carbonic anhydrase inhibitors, 'Daranide' in high doses causes some

decrease in renal blood flow and glomerular filtration rate.

INDICATIONS

'Daranide' is indicated for oral treatment of various types of glaucoma:

chronic simple (wide angle) glaucoma
acute congestive (narrow angle) glaucoma
chronic congestive glaucoma
secondary glaucoma (acute phase)
preoperative control of intraocular tension of glaucoma

DOSAGE

The usual adult dosage range is from 25 to 50 mg. (½ to 1 tablet) once to three or four times daily. Dosage must be carefully adjusted to meet the requirements of the individual patient.

Therapy with 'Daranide' is usually most successful when employed in conjunction with miotics such as pilocarpine, physostigmine, 'Floropryl' (isoflurophate), or 'Carcholin' (carbachol). In acute congestive glaucoma, 'Daranide,' with miotics, should be administered promptly in an attempt to bring about rapid reduction of intraocular tension. If this is not quickly relieved, surgery may be mandatory. In chronic congestive glaucoma, surgery may be necessary if the intraocular tension is not brought successfully under control after an adequate trial period of therapy with 'Daranide'.

PRECAUTIONS

Since 'Daranide' is a carbonic anhydrase inhibitor, certain side effects characteristic of this class of compounds will occur, particularly in the higher dosage ranges. These effects may in-

clude anorexia, nausea, vomiting, nervousness, lassitude, depression, confusion, disorientation, sedation, dizziness, ataxia, tremor, tinnitus, paresthesias, and pruritus. Fortunately, the therapeutic amount of 'Daranide' required for most patients is well beneath the level that will produce these effects. When they do occur, reduction of dosage or temporary discontinuance of the drug is indicated.

It is essential to keep a close check on the electrolyte balance of the patient during therapy with 'Daranide', as with patients on any other carbonic anhydrase inhibitor.

SUPPLIED

'Daranide' Tablets, 50 mg., bottles of 100 and 1000.

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AMERICAN JOURNAL OF OPHTHALMOLOGY
SERIES 3 · VOLUME 46 · NUMBER 3, PART I · SEPTEMBER, 1958

CONTENTS

AMERICAN JOURNAL OF OPHTHALMOLOGY

VOLUME 46 SEPTEMBER, 1958 NUMBER 3, PART I

HORNER'S SYNDROME: AN ANALYSIS OF 216 CASES*

CONRAD L. GILES, M.D., AND JOHN WOODWORTH HENDERSON, M.D.
Ann Arbor, Michigan

Although clinicians have recognized the symptom-complex comprising Horner's syndrome for almost a century, the relative frequency of lesions causing the syndrome has had inadequate study, either from the standpoint of etiologic agents or by anatomic location. A thorough review of the literature reveals many case reports, some with detailed descriptions of unique conditions which may result in a Horner's syndrome, but a detailed analysis of a significant series of cases is lacking.[1-10] For this reason the case records coded under this diagnosis at the University Hospital from January, 1936, to January, 1957, a 21-year period, were analyzed in order to study the location as well as the etiology of the lesions evoking this symptom-complex.

HISTORICAL DATA

The syndrome under discussion was first noted experimentally in 1852 by Claude Bernard,[11] but the first clinical description appeared in 1863 when three Americans—Weir-Mitchell, Morehouse, and Keen—found a right ptosis, miosis, and facial anhidrosis in a young soldier who had sustained a gunshot injury to the right side of his neck. These authors hypothesized in their book, *Gunshot Wounds and Other Nerve Injuries*,[12] that these clinical findings were due to interruption of the cervical sympathetics. Six years later, in 1869, the Swiss ophthalmologist, Johann Friedrich Horner, described the findings now associated with the syndrome bearing his name.[13]

CLINICAL DESCRIPTION

There is confusion in the literature in the designation of a given symptom-complex as a Horner's syndrome. The classical description lists the components as miosis, ptosis of the upper lid, anhidrosis, and enophthalmos—all on the affected side. It is evident, however, that clinicians have designated as Horner's syndrome many cases where only ptosis and miosis have been present. The pupillary miosis, dependent upon loss of sympathetic innervation to the dilator fibers of the iris, is the most constant finding and is reported to be most severe if the lower cervical or upper thoracic roots are sectioned. Loss of the pupillary reactions to light and accommodation does not occur, but these responses may be diminished.[14] Narrowing of the palpebral fissure occurs on the affected side from loss of sympathetic innervation to the smooth muscle of the lids. Loss of tone of Müller's muscle in the upper lid produces lowering of the upper lid border, and elevation of the lower lid border adds to the narrowing of the palpebral fissure as well. Anhidrosis of the homolateral face and neck occurs with sympathetic interruption unless the lesion is in the third neuron distal to the internal carotid plexus. This stems from the fact that the sweating fibers leave the system with the external carotid artery above the carotid bifurcation. For this reason anhidrosis is not a constant finding in what clinically may be denoted as a Horner's syndrome.

* From the Department of Ophthalmology, University of Michigan.

The enophthalmos is usually apparent rather than real, and is based upon narrowing of the palpebral fissure. Occasionally, for reasons not yet fully understood, it may be real and measurable.[15]

In addition to the basic signs just noted other features may sometimes accompany the syndrome. Heterochromia iridis has been reported as a part of the picture in cases where sympathetic interruption occurred before the age of two years.[16] A transitory rise in the temperature of the face on the homolateral side has been thought due to the loss of vasomotor control and dilatation of the blood vessels, to be followed by a temperature decrease and pallor. An alteration in tear secretion, either a decrease[17] or an increase,[18] has been reported less frequently. In cats, after pre- or postganglionic sympathectomy, a transitory fall in intraocular pressure of five to seven millimeters of mercury has been noted. This is followed by a return to normal or a slight increase in pressure after two days.[19] Cataract formation,[20] facial hemiatrophy, and an increase in the amplitude of accommodation have also been found associated with Horner's syndrome.[6, 21]

According to the strict definition of a Horner's syndrome, the classical components which result from interruption of the cervical sympathetic trunk are ptosis, miosis, anhidrosis, and enophthalmos. However, more detailed knowledge of the sympathetic nerve supply together with clinical usage has allowed the inclusion of any clinical interruption of the sympathetics to the eye as a Horner's syndrome.

Anatomy

The interruption of the sympathetic pathways of the autonomic nervous system at any point in their course from the hypothalamus to the orbit results in Horner's syndrome. This three-neuron pathway is entirely uncrossed and the fibers of the first neuron are generally believed to originate in the posterior portion of the hypothalamus

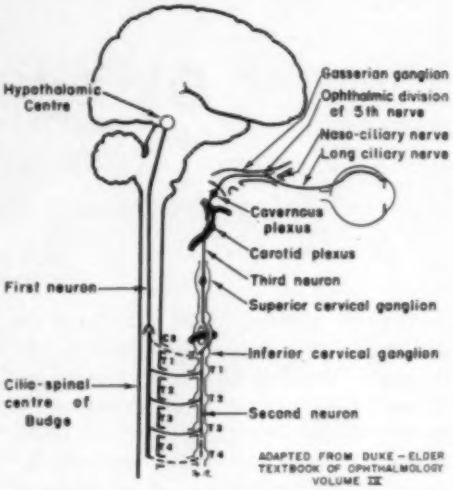

Fig. 1 (Giles and Henderson). Anatomic diagram.

(fig. 1). They then pass caudally through the tegmentum of the midbrain continuing in the reticular substance of the pons, medulla, and upper spinal cord to end in the anterolateral column of the lower cervical and upper thoracic portion of the spinal cord. Somewhere in the region between C7 and T4 a synapse occurs and the fibers of the second neuron leave the cord in the anterior roots of the lower cervical (C7-8) or upper thoracic (T1-4) nerves. By way of the white rami communicantes the fibers then pass to the upper thoracic and cervical sympathetic chain, ascending finally in the neck to the superior cervical ganglion, where another synapse occurs. From the superior cervical ganglion the third neuron, the sympathetic postganglionic fibers, forms the internal carotid plexus. According to Kuntz[22] they follow the internal carotid artery for a short distance and then deviate into the middle ear with the caroticotympanic fibers. Leaving the middle ear, they pass through the base of the cranium lateral to the nerve of the pterygoid canal and become associated with the cavernous plexus. These postganglionic fibers pass over the Gasserian ganglion and accompany the nasociliary nerve

into the orbit, then continue in the long ciliary branches of the nasociliary to penetrate the sclera medial and lateral to the optic nerve, passing forward to the plexus over the ciliary muscle. Here they innervate the dilator muscle of the iris, and in all probability the ciliary muscle as well.[21]

It is not mentioned in the literature whether the course of the sympathetic nerves to Müller's muscle of the lid or to the small amount of orbital smooth muscle differs from that of the pupillary fibers until they enter the orbit, but there is little reason to expect a difference.

As noted in the clinical description of the syndrome, the fibers innervating the sweat glands of the face and neck travel with the pupillary fibers up to the bifurcation of the common carotid artery. Beyond this point the sweating fibers course with the external carotid distribution to the face.

Case material

The purpose of this study was to determine as nearly as possible the anatomic site of sympathetic interruption and the etiologic factors involved in a series of cases with Horner's syndrome. Two hundred sixteen cases were found coded in the University Hospital records in the 21 years between January, 1936, and January, 1957. In all the records the diagnosis of Horner's syndrome had been made or verified either by the neurologic or ophthalmologic service. In all cases the constant diagnostic features included ptosis and miosis plus a variable number of other signs. As is the case in any series of this sort, the conclusion as to location of the lesion must often be inferred from history, physical and neurologic findings, as well as surgical reports, since in many a necropsy was not performed.

It is likely that the cases selected from the coding system do not necessarily represent the total encountered in the hospital during this period. Further, the nature of selection of cases in any large diagnostic center does not necessarily reflect the incidence in the population at large, since the cases are preselected by the very fact of referral.

Levels of interruption

Table 1 lists the anatomic location of the lesions producing Horner's syndrome. The majority of instances were due to interruption in the pathways distal to the spinal cord and proximal to the intracerebral portion of the postganglionics. Of the 216 cases 101, or 46.8 percent, were due to involvement of the cervical sympathetic trunk either in its preganglionic fibers or in the postanglionics prior to entering the cranium. Interruption of the sympathetic fibers in the region of the lower cervical and upper thoracic anterior spinal roots was found in 43 cases, or 19.9 percent. In only 19 cases involvement of the descending intramedullary first neuron was causative; in 12, or 5.6 percent, the lesion was located in the brain stem, and in seven or 3.2 percent, interruption occurred in the spinal cord. Statements by many authors have indicated that interruption of the third neuron intracranially is uncommon and the present series bears this out. In only one case (0.4 percent of the series) was an intracranial postganglionic lesion implicated. It was impossible to localize the site of the lesion in 52, or almost 25 percent of the cases. Although a few of these patients were examined thoroughly, the majority were noted as congenital in nature without further complete investigation.

TABLE 1

Location of lesion

	No.	Percent
Sympathetic trunk	101	46.8
Low cervical-High thoracic ant. spinal roots	43	19.9
Brain stem	12	5.6
Spinal cord	7	3.2
Intracerebral postganglionics	1	0.4
Undetermined	52	24.1
Total	216	100.0

TABLE 2

ETIOLOGY OF LESION

			No.	Percent
Neoplasia	Benign	19	77	35.6
	Malignant	58		
Surgical procedures	Primary	19	40	18.5
	Secondary	21		
Trauma	Birth	12	28	13.0
	Other	16		
Vascular disease			10	4.6
Other			9	4.2
Undetermined			52	24.1
Total			216	100.0

TABLE 3

NEOPLASMS CAUSING HORNER'S SYNDROME

Benign		
Thyroid adenoma		12
Neurofibroma		7
Total		19
Malignant		
Bronchogenic carcinoma		27
Metastatic carcinoma		19
Breast	8	
Nasopharynx	3	
Colon	1	
Tonsil	1	
Larynx	1	
Cervix	1	
? Primary	4	
Metastatic sarcoma		4
Hodgkin's disease		3
Thyroid carcinoma		2
Primary sarcoma		1
Lymphoblastoma		1
Histology unknown		1
Total		58

ETIOLOGY

Several excellent reviews[1, 2, 14] have been written dealing with the various factors involved in the production of Horner's syndrome. Although literally hundreds of agents have been incriminated, there has been no report of the relative frequency of causes in a series of patients. Table 2 summarizes our findings. The most common factor producing the syndrome was neoplasia, more often malignant than benign. Neoplasms were responsible in 77 cases, or 35.6 percent; of these 58 were malignant and 19 benign. Bronchogenic carcinoma, usually of the upper lobes, producing a Horner's syndrome by pressure on the cervical sympathetic trunks or by pressure on or invasion of the brachial plexus, was the most common entity. Metastatic carcinoma of the breast, Hodgkin's disease, carcinoma of the thyroid, sarcoma, and metastatic carcinoma in general were other common malignancies noted in this series. Benign neoplasms of importance included large thyroid adenomas and neurofibromata.

Table 3 lists the frequency of the various neoplasms found in the series. In all but 10 cases these neoplasms caused a Horner's syndrome by interruption of the sympathetic pathways in the sympathetic trunk or in the postganglionic fibers prior to their intracranial course. These 10 exceptions are listed in Table 4.

Surgical interference with the sympathetics accounted for 18.5 percent of the cases in the series (table 2). The patients have been grouped as "primary" or "secondary" according to whether a Horner's syndrome might reasonably be expected as a result of the surgery. In the "primary" group of 19 cases were 10 patients with an upper dorsal and/or lower cervical sympathectomy, eight with stellate ganglion blocks, and one whose Horner's syndrome resulted from a cordot-

TABLE 4

NEOPLASMS IN SITES OTHER THAN SYMPATHETIC TRUNK AND POSTGANGLIONIC FIBERS

Anterior spinal roots		
Benign		
Neurofibroma		4
Malignant		
Bronchogenic carcinoma		3
Metastatic carcinoma		2
Breast	1	
Cervix	1	
Spinal cord		
Malignant		
Histology unknown		1
Total		10

omy. "Secondary" cases (21 in number) included thyroidectomy (four cases), phrenicectomy (six cases), and tumor excision (11 cases).

Nonoperative truma was the etiologic agent in 13 percent, or 28 cases, of the series. Brachial plexus injuries, due either to birth trauma or to automobile accidents, made up the largest segment of this group, with gunshot wounds accounting for only two cases. Vascular disease of the brain stem, either thrombotic or embolic, elicited a Horner's syndrome in 10 patients (4.6 percent).

Other etiologies noted in the series were multiple sclerosis, caudal analgesia (two cases), congenital malformation, encephalitis, tuberculosis, chronic infective granuloma, syringomyelia, tabes dorsalis, and herpes zoster. In the remaining 52 cases no etiologic agent was found.

TABLE 5

ETIOLOGY BY LOCATION

1. *Sympathetic trunk* (*101 cases*)		No.	Percent
Neoplasia			
Benign	15		
Malignant	52	67	66.5
Surgical procedures			
Primary	18		
Secondary	13	31	30.7
Trauma (none caused by birth)		2	1.9
Other		1	0.9
Total		101	100.0

ETIOLOGY BY LOCATION

In order to gain the proper perspective of the etiology and location of lesions causing Horner's syndrome it is helpful to examine the relative frequency of occurrence of the etiologic agent at the specific site of involvement.

At the most common site of involvement, the cervical sympathetic trunk including the postganglionic fibers proximal to their intracerebral course (table 5), neoplasia was the predominant etiologic factor in the production of Horner's syndrome. Sixty-seven of the 101 cases showing such involvement of the sympathetic trunk were due to neoplasms (66.5 percent). Malignant tumors were present in 52 of the 67 cases and benign in 15. The authors found it difficult to determine the specific site of involvement in pulmonary tumors. The problem was to decide whether the production of a Horner's syndrome due to a tumor of the upper lobes was the result of pressure upon or invasion of the brachial plexus, or due to pressure upon the cervical sympathetic trunk. In general the criterion used to indicate implication of the brachial plexus and the anterior roots of the spinal nerves in this area was a combination of one or more of the associated signs and symptoms of weakness, atrophy, radicular pain, and sensory loss in the distribution of the roots involved. If none of these criteria was present it was considered likely that the Horner's syndrome was the result of pressure on or direct involvement of the cervical sympathetic trunk. Where the syndrome was found due to involvement of the sympathetic trunk, 31 cases (or 30.7 percent) were the result of surgical procedures. Eighteen of these were of the so-called "primary" type and 13 were "secondary." Traumatic injuries accounted for two cases, and a chronic infective granuloma with its resulting lymphadenopathy was causative in the remaining patient.

Analysis of those lesions located in the low cervical and high thoracic anterior spinal roots (table 6) indicated that in 23 out of 43 cases (or 53.6 percent of the time) a traumatic injury was the cause. In only nine patients (20.9 percent of the 43) neoplasia was the cause (benign tumors in five and malignancies in four—see Table 4). In eight cases (18.6 percent of 43) surgical procedures were incriminated—none of these directed primarily toward the sympathetics. The remaining three cases were due to high epidural blocks used in obstetrical procedures (two cases) and to

TABLE 6

ETIOLOGY BY LOCATION

2. *Low cervical-high thoracic anterior spinal roots (43 cases)*		No.	Percent
Neoplasia			
Benign	4		
Malignant	5	9	20.9
Surgical procedures (all secondary)		8	18.6
Trauma			
Birth	12		
Other	11	23	53.6
Other		3	6.9
Total		43	100.0

a congenital malformation of the cervical vertebrae (one case).

Posterior inferior cerebellar artery thrombosis and superior cerebellar artery thrombosis were the cause in 10 of the 12 cases where a Horner's syndrome was due to interruption of pathways in the brain stem (table 7). The other two etiologic factors were multiple sclerosis and encephalitis, each occurring in one patient.

The least affected area proximal to the postganglionic fibers, the spinal cord (table 7), showed a wide range of etiologies. Trauma caused three cases while a malignant cord tumor, tabes dorsalis, syringomyelia, and a cordotomy each accounted for one case.

In only one case of the entire series of 216 was it possible to localize with certainty a lesion occurring in the intracerebral portion of the postganglionic pathway. In this patient a Horner's syndrome occurred in association with herpes zoster ophthalmicus, clearing when the herpes resolved. Several authors have implicated such entities as tumors of the Gasserian ganglion, orbital tumors, intracranial internal carotid aneurysms, to name a few. According to our series, such cases must indeed be unusual.

It has been generally recognized that the majority of lesions causing Horner's syndrome involve the second neuron.[2] This is substantiated by the present study. Over two thirds of the cases were the result of interruption of the second neuron, or the extracranial portion of the third neuron. No attempt was made to differentiate lesions in the neck as to location proximal or distal to the superior cervical ganglion because of inadequate diagnostic criteria.

TABLE 7

ETIOLOGY BY LOCATION

3. *Brain stem (12 cases)*	No.	Percent
Vascular	10	83.3
Other	2	16.7
Total	12	100.0

4. *Spinal cord (7 cases)*	No.	Percent
Neoplasm (malignant)	1	14.3
Surgical procedure (primary)	1	14.3
Trauma (none at birth)	3	42.9
Other	2	28.5
Total	7	100.0

ETIOLOGY BY AGE

One of the simplest historical facts, age at the time of onset, may be a most important diagnostic aid to the clinician investigating a Horner's syndrome of unknown etiology. Table 8 summarizes the frequency of the various etiologic agents by age groups. In the younger age group, from birth to 20 years of age, trauma was the cause in 20 of

TABLE 8

ETIOLOGY BY AGE GROUP

	Birth-20 Years	21-50 Years	51 Years and Over
Neoplasia	4 cases	33 cases	40 cases
(Benign)	(0)	(16)	(3)
(Malignant)	(4)	(17)	(37)
Surgical procedures	7 cases	20 cases	13 cases
(Primary)	(3)	(8)	(8)
(Secondary)	(4)	(12)	(5)
Trauma	20 cases	6 cases	2 cases
(Birth)	(12)	(0)	(0)
(Other)	(8)	(6)	(2)
Vascular disease	0 cases	4 cases	6 cases
Other	1 case	6 cases	2 cases
Totals	32 cases	69 cases	63 cases

the 32 cases. Birth trauma was responsible for 12, automobile accidents and gunshot injuries for eight cases, while neoplasia and surgical procedures were of less importance, producing only four and seven cases respectively.

Sixty-nine cases were found in the group from 21 to 50 years of age. Almost half of them (33) were the result of neoplasms. Benign tumors produced 16 cases while malignant tumors caused 17. Surgical operations accounted for 20, 12 being "secondary" and eight "primary" procedures. Vascular disease involved four cases in this age group, while trauma produced six.

Sixty-three cases were found in individuals 51 years of age and older. Again in this age group (as in the group from 21 to 50 years) neoplasia was the most important factor. However, in this oldest group, malignant tumors were preponderant (37 cases) while benign tumors resulted in only three instances of Horner's syndrome. Surgical procedures were involved in 13 patients, vascular disease in six, and trauma elicited the syndrome in only two.

Of the total series of 164 localizable lesions, 20 percent occurred in the first two decades of life, another 40 percent in the next three decades and the final 40 percent in the group over 50. This indicates that birth trauma should be thought of first in the group under 20 years of age, while neoplasia (benign or malignant) should be considered immediately in a patient between the ages of 21 and 50 presenting with a Horner's syndrome. In the group over 50 years malignant neoplasms must be ruled out first.

Clinical application

The practical significance of this study lies in its application to the clinical investigation of the syndrome. The importance of identifying the level of involvement together with the etiologic factor cannot be overemphasized since early detection of the cause and its correction may prove to be life-saving. Certain basic tools and several specialized procedures are available to the clinician in his investigation.

The single most important means of investigation is a detailed history. Emphasis should be placed on detailed reporting of all operative procedures, head or neck trauma, and the age and time of onset of the Horner's syndrome.

In addition to complete physical examination, a detailed neurologic investigation is of importance in determining the association of other neurologic deficits.

Stereoscopic and lateral views of the chest, routine skull films, and lateral cervical and upper thoracic vertebral views should be considered as routine studies in the evaluation of an obscure Horner's syndrome.

In instances where there is a reasonable suspicion of the possibility of a spinal cord tumor, myelography should be performed.

A detailed ophthalmologic examination is necessary. This should include pharmacologic testing of the pupil. The presence or absence of heterochromia iridis can be important in determining whether or not the Horner's syndrome was present before the age of two years.

Sweat testing should be employed if observation does not reveal a definite anihidrosis. The color change of Quinizarin Compound® from black to purple on the clean face of the patient indicates the presence of sweating. As noted earlier, the absence of anhidrosis in the presence of a ptosis and miosis localizes the lesion distal to the carotid artery bifurcation.

Summary and Conclusions

The 216 cases of Horner's syndrome seen in the University Hospital from January, 1936, until January, 1957, are analyzed with respect to location and etiology of the lesion eliciting the syndrome. A brief review of the neuroanatomic pathways is presented, together with an outline for clinical investigation.

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The following conclusions can be drawn from this study:

1. The most common site of interruption of the sympathetic pathway to the eye is in the sympathetic trunk and in the postganglionic fibers before they course intracranially. The upper thoracic and lower cervical anterior spinal roots are affected less than half as often as the sympathetic trunk, while the brain stem, spinal cord, and intracerebral postganglionics are only rarely affected.

2. Neoplasia, malignant far more often than benign, is the most common etiologic agent. Bronchogenic and metastatic carcinoma are the most frequent malignancies, and neurofibroma and thyroid adenoma are the only significant benign tumors. Surgical procedures, nonoperative trauma, and vascular disease are less important causes of Horner's syndrome.

3. Neoplasia is the most important factor incriminated in sympathetic trunk lesions while trauma and vascular disease are the most frequently encountered agents at the anterior spinal root and brain stem levels respectively. Spinal cord sympathetic pathway interruption was most commonly a result of trauma. Herpes zoster was the only lesion implicated in an intracranial postganglionic lesion.

4. Horner's syndrome is seen most commonly in the older age groups. In the first two decades of life trauma is the most common etiology. Malignant and benign neoplasms account for the largest percentage of sympathetic interruption in the third, fourth, and fifth decades. In the group over 50 years of age malignant tumors are more likely.

5. In view of the frequently serious nature of the lesions involved in the production of a Horner's syndrome, a complete investigation is warranted.

University Hospital.

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FUCHS' EPITHELIAL DYSTROPHY OF THE CORNEA*

A CLINICAL AND HISTOPATHOLOGIC STUDY

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In 1910, soon after Fuchs[1] had described the clinical picture of the corneal disease named after him, he obtained a piece of superficial cornea from an advanced case and added a histopathologic study of the epithelial lesions. With the available material he could not, however, elucidate their pathogenesis.

It was through biomicroscopy that our present concept of this dystrophy as a combination of endothelial and epithelial disease was established during the following 20 years (Vogt,[2] Friedenwald,[3] Kraupa,[4] et al.).

A recent report by Wolter, Henderson, and Gates[5] of the histopathologic findings in a corneal button, excised during a penetrating keratoplasty in such a case and studied by means of del Rio Hortega's silver carbonate technique, has added valuable information to our scanty knowledge of the pathologic processes in this condition. These authors demonstrated an extensive destruction of the stromal and epithelial nerves, thus explaining the hypo- and insensitivity of the cornea in this disease mentioned repeatedly in the literature.

An entire bulb of an apparently unilateral case was studied by v. Hippel.[6] In this eye the disease had run an unusual, short course of six months. Clinically, there had been an extensive bullous keratopathy and some thickening of the central corneal stroma. Intraocular tension was low and the corneal sensitivity was diminished. The sensitivity of the other eye was not tested and no examination with the slitlamp of the corneal endothelium in either eye was made, so that a bilateral dystrophy, at an early stage in the fellow eye, cannot be entirely ruled out. There was a chronic purulent dacryocystitis on the side of the obvious dystrophy.

v. Hippel found the histopathologic changes limited to the corneal endothelium and epithelium. Descemet's and Bowman's membranes and the corneal stroma were normal. There was extreme rarefaction of the endothelium without any evidence of hyperactivity resulting in excrescences of Descemet's membrane.

CASE REPORT

The following histopathologic study was made on the left eye of a woman, 61 years of age, who had been under observation with a bilateral endothelial and epithelial dystrophy for 25 years before one of the eyes had to be excised.

HISTORY

When first seen in 1929 at the age of 37 years, this woman gave a history of intermittent redness and pain in her left eye for the past year, which had started soon after the splashing of some lye soap into it. Up to that time vision in the two eyes had been equal.

At the time of her first visit the right vision was 20/25; the left vision, 5/200. In both eyes there was an extreme degree of cornea guttata and the right eye showed a typical picture of epithelial dystrophy (Fuchs). In the left cornea there was dense disciform central clouding and a few precipitates were applied to the posterior corneal surface. Corneal sensitivity was markedly reduced in the right eye, absent in the left.

General medical examination revealed mild obesity, hypertrophic arthritis, a blood pressure of 140/70, and an empyema of the

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left antrum. The latter was cleared up after several weeks of therapy. A year later a tonsillectomy was performed.

The left eye became quiescent under local treatment with atropine, and vision improved to 10/200. During the following years there were repeated attacks of pain and redness in this eye, sometimes with small deep infiltrates within or in the vicinity of the central disc, other times with tiny superficial ulcers.

During a quiescent period between 1939 and 1941 the left vision could be improved to 20/40 with a correction of +6.0D. sph. ⌒ +200D. cyl. ax. 170°. The right vision, with a correction of +1.5D. sph. ⌒ +0.5D. cyl. ax. 110° was still 20/25. Both fundi were normal.

After 1941 repeated superficial and deep infiltrates of the left cornea, always accompanied by a mild iritis, recurred at more frequent intervals, and in 1953 a central corneal ectasia developed which led to the enucleation. The right vision at that time was 20/30. No increase of intraocular tension had ever been found in either eye.

In 1955, the right vision was reduced to 20/100 improvable to 20/60 with diamox, which, however, was not tolerated by the patient. There have been no attacks of pain or redness in this eye and at the present time vision is reduced to 10/200, mainly due to a nuclear type of cataract. Surgical therapy of any kind has been refused by the patient.

Histopathologic findings

Half of the globe was embedded in paraffin for special stains, the other half in celloidin for serial sections.

The center of the cornea was thin and ectatic. Bowman's and Descemet's membranes were completely destroyed within this central area which consisted of a loose network of proliferated keratoblasts with inclusions of necrotic debris. These sites of focal necrosis were surrounded and infiltrated by polymorphonuclear leukocytes. Some of these small abscesses contained fragments of Descemet's membrane, especially along an area which suggested seepage (fig. 1). This area at the superior nasal edge of the ectasia was indicated anteriorly by a short ingrowth of the epithelium, and posteriorly by the frayed endings of Descemet's membrane and a drawn out anterior synechia at one point. In the iris this point of adhesion was marked by a mass of Descemet-like substance, indicating that the contact between iris and cornea had existed there for some time.

The stroma adjoining the ectatic portion contained multiple small deep abscesses around the splitting ends of Descemet's membrane (fig. 2) and in its middle layers. The anterior layers showed an increased number of keratoblasts especially underneath the irregularly thickened epithelium where Bowman's membrane was destroyed.

The most peripheral stroma contained a few new vessels which had grown through it toward the central lesion, but otherwise appeared to be normal, with normal metachromatic staining property (toluidine blue).

The epithelium was abnormal almost throughout. The peripheral and pericentral portions showed the dystrophy of the original epithelium, while over the central areas an abnormally thick or abnormally thin epithelium had grown over formerly denuded places. The dystrophy (figs. 3, 4, 5, and 6) was indicated by great variations in the staining property of the cells, the foot cells being unusually large, pale, and irregular in size, and the superficial layers being flat, with dark, shrunken nuclei. Periodic acid Schiff stain indicated almost complete absence of the basement membrane in the pericentral and central regions.

Within a broad pericentral zone there were numerous interruptions and gaps of Bowman's membrane whose tapered edges characterized them as preformed nerve canals (figs. 3 and 4), now widened and containing a scant number of nuclei belonging to proliferated Schwann cells (Salz-

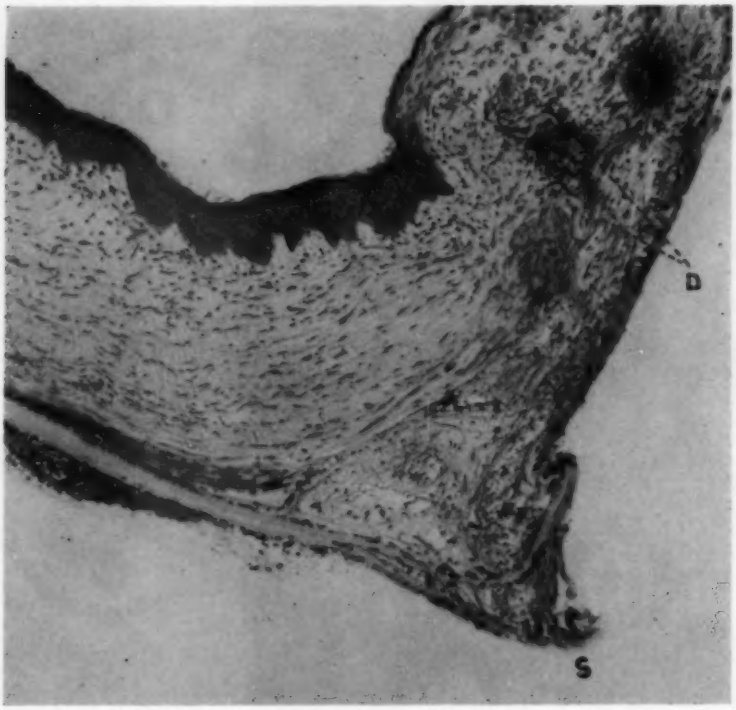

Fig. 1 (Klien). Fraying and rupture of Descemet's membrane at edge of corneal ectasia, small abscesses in various layers of corneal stroma, some containing fragments of Descemet's membrane (D). (S) Point of torn anterior synechia. (Hematoxylin-eosin, ×105.)

mann[8]). No proliferation of keratoblasts was discernible in these areas. In the immediate neighborhood of some of these conspicuous, oblique nerve canals thin fibrous membranes originated, separating the epithelium from Bowman's membrane over more or less extensive stretches (figs. 3 and 5). Bowman's membrane, of normal thickness in the periphery, became gradually thinner toward the center (fig. 6), showing an increasing number of dehiscences, until it disappeared entirely over the central disc of maximally damaged stroma.

Descemet's membrane had normal width in the extreme periphery but in the pericentral region attained a thickness of four to five times its normal diameter, with a large number of warty excrescences of different sizes. The largest of these warts seemed to interrupt the continuity of the endothelial lining. The nuclei of the endothelial cells were lying in the valleys between the excrescences, irregularly spaced, depending upon the latter's sizes (fig. 7).

Many of the endothelial cells contained pigmented granules some of which gave a positive periodic acid Schiff stain indicating a lipopigment. Similar granules were contained within some of the endotheliol cells of the anterior surface of the iris. It is not known whether or not this pigmentation is a characteristic part of the endothelial dystrophy as has been suggested by some authors from clinical observations (Kraupa,[4] Vogt,[2] Friedenwald[3]).

The angle of the anterior chamber was

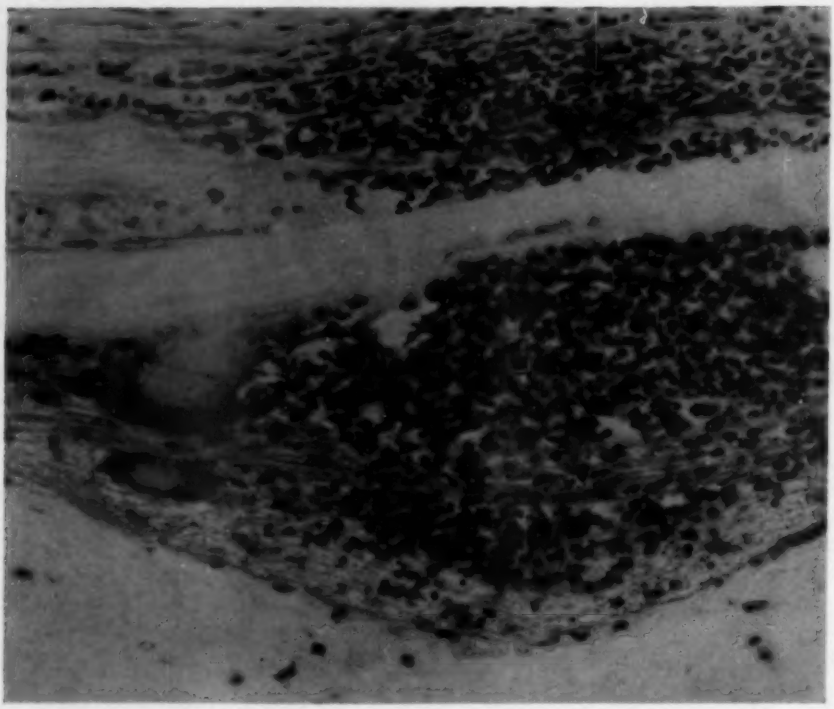

Fig. 2 (Klien). Circumscribed accumulation of polymorphonuclear leukocytes with group of giant cells on each side of Descemet's membrane. (Hematoxylin-eosin, ×440.)

Fig. 3 (Klien). Early widening of nerve canal in Bowman's membrane. Note tapered edges of Bowman's membrane. Incipient proliferation of Schwann cells in and in front of Bowman's membrane. Stroma normal. Degeneration of foot cells of epithelium. (Hematoxylin-eosin, ×575.)

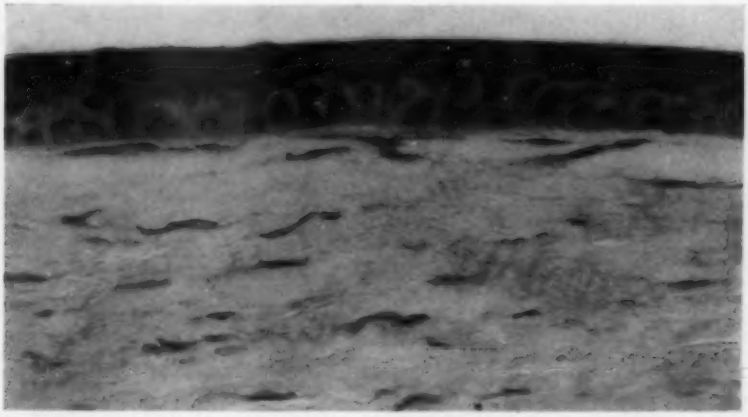

Fig. 4 (Klien). Two widened Bowman canals with proliferation of Schwann cells. Thinning of fragmented piece of Bowman's membrane. (Hematoxylin-eosin, ×425.)

loosely filled with a mixture of lymphocytes, monocytes, polymorphonuclear leucocytes, fibrin, and a small number of erythrocytes. A very short peripheral anterior synechia had formed in several places.

The pupillary portion of the iris was considerably atrophic, especially where it was drawn toward the posterior corneal surface. The stroma of iris and corona ciliaris was moderately infiltrated with lymphocytes and plasma cells. The lens was in situ. The posterior segment of the eye was normal.

Comment

Tremendous thickening of Descemet's membrane such as encountered in this case may not be an integral part of the picture of advanced endothelial and epithelial dystrophy, but in this instance may have developed in response to the slowly progressing weakness and deformity of the corneal stroma. At any rate, it permitted the conclusion that even in an advanced dystrophy some endothelium is present and capable of physiologic activity or hyperactivity.

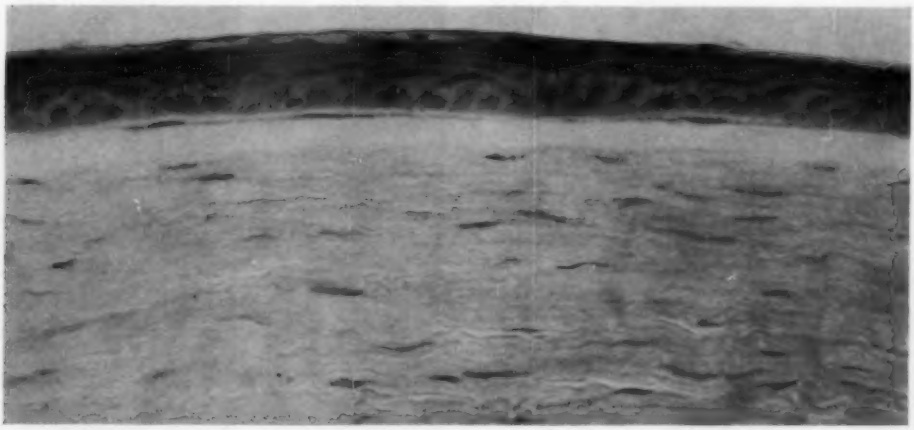

Fig. 5 (Klien). Inconspicuous but extensive membrane of flat cells originating at widened Bowman canals (Schwann cells) separating epithelium from Bowman's membrane. Irregularities of epithelial cells, especially foot cells. (Hematoxylin-eosin, ×425.)

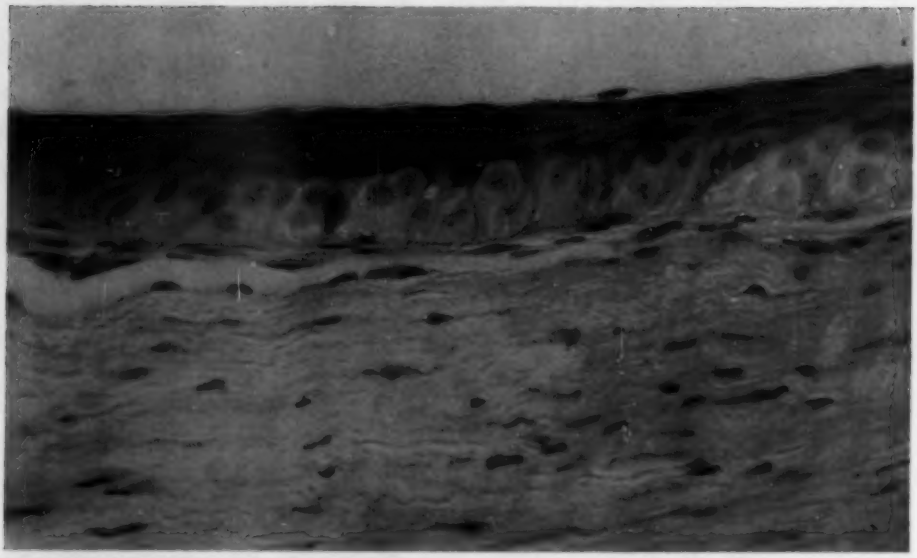

Fig. 6 (Klien). Extreme thinning of Bowman's membrane in immediate neighborhood of central degeneration. Proliferation of keratoblasts in stroma underneath it. (Hematoxylin-eosin, ×500.)

The clinical picture of extreme cornea guttata which usually precedes the stromal and epithelial disease also suggests pathologic activity rather than destruction of the endothelium. By formation of the numerous excrescences the posterior corneal surface becomes considerably expanded and thus dehiscences in the endothelial layer develop. Gradual rarefaction of the endothelial cells on the basis of such mechanical insult appears to be more probable than a primary degeneration.

The excrescences of Descemet's membrane appear to be an integral part of the typical picture of the dystrophy while an additional diffuse thickening of the membrane may or may not be present. In Wolter, Henderson, and Gates' case of a fairly advanced dystrophy, it was absent. On the other hand, Goar[7] described a multifold thickening of Descemet's membrane in a case of endothelial dystrophy without epithelial or stromal changes.

The cause of the endothelial hyperactivity is entirely unknown. It could be protective in nature against injurious metabolites in the aqueous which might damage the cornea more rapidly without it. In most cases of cornea guttata and epithelial dystrophy, the

Fig. 7 (Klien). Tremendous thickening of Descemet's membrane with excrescences on posterior surface. Incipient dehiscences of endothelial lining. (Hematoxylin-eosin, ×550.)

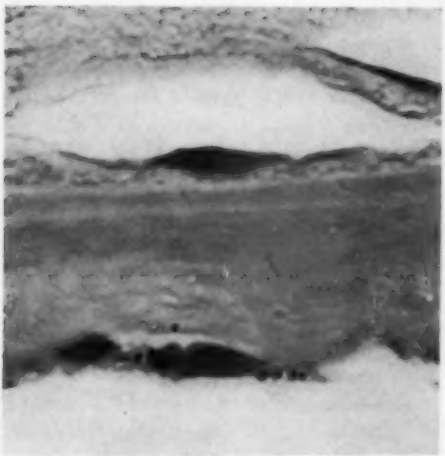

Fig. 8 (Klien). Endothelial cell in valley between excrescences of Descemet's membrane, containing pigment granules. (Hematoxylin-eosin, ×980.)

course of the disease is protracted over many decades. In an exceptional case it may be accelerated (v. Hippel) following a severe initial insult to the endothelium which results in its rapid destruction rather than in the more common irritation and hyperactivity with formation of new, Descemet-like substance.

The small corneal infiltrates, appearing and disappearing in all layers of the central cornea over a period of 24 years, were revealed by histopathologic study to be basically events comparable to those occurring on a larger scale in ring abscess of the cornea, namely minute sequestrations of stroma attracting polymorphonuclear leucocytes from the nearest vessels and thus forming small abscesses.

In this connection it is interesting that in the case of Wolter, Henderson, and Gates there was a history of ulceration about a year prior to the removal of the corneal button. Even after one year these authors still found some infiltration with polymorphonuclear leucocytes and lymphocytes in stroma and epithelium of the central area. There was no history of injury in their case but one wonders if minor trauma, even such as tonometry, might not produce sequestration of a small piece in an already badly damaged cornea and lead to a minute abscess or ulcer.

In our case the injury reported by the patient had not caused much immediate discomfort but was remembered again when the eye became troublesome. It apparently had precipitated the intermittent, extremely slow process of destruction and repair in the corneal stroma which characterized the course of the disease in this eye over a period of two decades and a half. During this time there was only one period of remission lasting about two years.

It is remarkable that during this period of remission the vision of this eye, which in other years varied from five to 10/200, could be improved to 20/40 with a correction which indicated an already considerable degree of corneal deformity. The explanation for this unusual improvement of vision may lie in the fact that the process of repair, still evident in the enucleated eye, consisted almost exclusively of proliferation of keratoblasts with formation of new lamellae, and of an additional thickening and ingrowth of epithelium in areas of superficial stromal destruction, both processes resulting in new tissue of relatively great transparency.

The large number of widened nerve canals through Bowman's membrane within a broad circular zone around the central area of stromal destruction deserves special emphasis. They seem to constitute the first visible manifestation of a disturbance in the corneal nerves in meridional sections. They are similar to those described by Salzmann[8] in chronic glaucoma and thought by him to be pathognomonic for this condition. The cells proliferating within these oblique canals appear to be not keratoblasts but Schwann cells, which eventually form a thin layer separating the epithelium from Bowman's membrane, at first in the immediate neighborhood of the canal, later over more extensive areas.

Since Salzmann's first description of this neural dystrophy in glaucoma it has also been found in keratoconus (Klien[9]). It is

possible that abnormal hydration with or without a slowly developing deformity of the cornea may play a role in its pathogenesis.

The proliferations of Schwann cells appear to undergo complete destruction later, together with that of Bowman's membrane with which they are intimately connected from the beginning; thus, only the reparative proliferation of keratoblasts is evident in areas of advanced dystrophy.

It would be interesting and possibly of some clinical importance to find out at what stage of the dystrophy the disturbance of the corneal sensitivity develops. Wolter, et al. are probably correct in assuming that damage to the corneal nerves is secondary to the stromal and epithelial involvement.

Summary and Conclusions

The clinical course of an eye with endothelial and epithelial dystrophy (Fuchs), observed with its similarly affected fellow eye for 24 years, and the histopathologic findings at the end of this period suggest as the basic pathologic process an insult to the endothelium. In a typical case, this results in an initial hyperactivity of these cells with formation of a variable amount of a Descemet-like substance, and later in their gradual rarefaction on a mechanical basis.

There is some evidence that in an exceptional case the insult, of as yet undetermined origin, may lead to immediate destruction rather than to irritation of the endothelium and to an atypical accelerated course of the disease, such as was reported by v. Hippel.

Abnormal hydration of the cornea leads to an intermittent process in the stroma of destruction and repair, which may be complicated by minute sequestrations (accelerated focal destruction) and abscess formations. These may occur under the influence of minor traumas, and in the end stages of the disease possibly also spontaneously.

The traumatic element should be avoided by extreme care during routine procedures such as tonometry, and by the wearing of protective lenses as soon as a disturbance of the corneal sensitivity can be demonstrated.

Corneal sensitivity should be tested at regular intervals in all cases of advanced cornea guttata to determine at what stage of the dystrophy its disturbance appears.

In case of surgical therapy with keratoplasty removal of a large button inclusive of the zone of neural dystrophy around the central lesion may be more rewarding than a small transplant.

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TONOGRAPHY AND ANGLE-CLOSURE GLAUCOMA: DIAGNOSIS AND THERAPY*

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INTRODUCTION

The increasing availability of tonography for the diagnosis and management of the glaucomas necessitates repeated evaluation of its usefulness. In angle-closure glaucoma, tonography and provocative tests may supplement the gonioscopic findings and history in establishing the diagnosis.[1-3] Preoperative tonography affords additional information as to the type of surgery required in eyes with this defect.[4] The present paper is concerned with tonographic findings in patients with angle-closure glaucoma studied over a three-year period (July 1, 1954, to June 30, 1957). Attention was directed specifically to (1) the value of the mydriasis provocative test combined with tonography as a means of detecting angle-closure; and to (2) the use of tonographic estimates of the adequacy of outflow channels as a guide to the type of surgery indicated (iridectomy or filtering operation).

METHODS

Tonography was carried out with an electronic tonometer connected to a Leeds and Northrup recorder. All tracings were interpreted on the basis of the 1955 Friedenwald tables[5] with suitable correction for episcleral venous pressure and scleral rigidity.[6]

For diagnostic purposes, patients referred with narrow chamber angles, but no evidence of inflammatory disease, surgery, or elevated intraocular pressure (Schiøtz scale reading less than 4.0 with a 5.5 gram weight), were subjected to an euphthalmine provocative test. This test consisted of (1) tonography while on no medication, (2) the administration of five-percent euphthalmine to one eye, and (3) repeat tonography after one hour. The test was carried out on 58 eyes with narrow angles and 24 eyes with normal angles.

Preoperative evaluation in patients who had had acute attacks of angle-closure or positive provocative tests was based upon single or multiple tonographic tracings while on medical therapy. Postoperatively the same eyes were evaluated as to the degree of pressure control, the need for medical therapy, the presence or absence of further acute attacks, the retention of vision and field, and the facility of outflow. There were 182 eyes of 110 patients which could be so studied before and after surgery. These were clinic and private patients of the resident and visiting staff of the Department of Ophthalmology, Washington University.

RESULTS

A. DIAGNOSIS (Table 1)

Of the 58 normotensive eyes with narrow angles subjected to the mydriasis provocative test, 16 (28 percent)were found to have a rise in intraocular pressure of 8.0 mm.Hg or more. However, 39 (67 percent) of these same 58 eyes demonstrated a decrease in outflow facility of more than 25 percent. Of the 58 eyes, 32 presented reasonable clinical evidence of being capable of angle-closure. This diagnosis was based upon a subsequent occurrence of an attack

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TABLE 1
TONOGRAPHY AND THE DIAGNOSIS OF ANGLE-CLOSURE GLAUCOMA: THE EUPHTHALMINE PROVOCATIVE TEST

Gonioscopy	Number of Eyes	Incidence of Increase Intraocular Pressure (over 8.0 mm. Hg)	Incidence of Decrease Outflow Facility (more than 25%)
Narrow angles			
Closure*	32	14 (44%)	28 (88%)
No closure	26	2 (8%)	11 (42%)
Total	58	16 (28%)	39 (67%)
Normal angles	24	0 (0%)	0 (0%)

* Subsequently in same eye or history of closure in contralateral eye.

of angle-closure in this eye or a history of closure in the contralateral eye. Of this group, mydriasis induced a rise in intraocular pressure of 8.0 mm.Hg or more in only 14 (44 percent). However, 28 (88 percent) of these same eyes decreased their outflow facility by more than 25 percent. None of the 24 eyes with normal angles had either a pressure rise or a significant decrease in outflow facility.

B. Therapy

In the 110 patients (75 females and 35 males) available for this study, 209 operations were performed on 182 eyes. These consisted of 100 peripheral iridectomies (all initial procedures) and 109 iridencleises (82 as the primary surgery and 27 reoperations). On the basis of an evaluation of the results of surgery, the eyes were divided into three categories: (1) no further attacks and adequate pressure control at all subsequent measurements without any medication; (2) similar control but requiring medication (miotics ± carbonic anhydrase inhibitors); and (3) failure of control in spite of the use of miotics and carbonic anhydrase inhibitors. (Many eyes in category 3 were subjected to reoperation.)

The over-all prognosis for the entire series is presented in these terms in Table 2. Of the 182 eyes, 109 (60 percent) could be controlled without medication after an initial procedure, 41 (23 percent) required medical therapy for continued control, and 32 (17 percent) failed to be controlled by the initial operation and subsequent medical therapy. After reoperation of 27 of these 32 failures, 16 additional eyes were successfully controlled. Thus, in the entire series, only 16 (nine percent) of eyes with angle-closure glaucoma failed to be controlled by surgical and medical means.

The results of peripheral iridectomy and iridencleisis are not very different when viewed in the manner presented in Table 2. However when eyes are classified on the basis of preoperative tonographic findings, striking differences appear. In Table 3 the 182 eyes are divided into four groups on the basis of the preoperative outflow facility. When analyzed in this fashion the results of initial surgery vary from a three-percent

TABLE 2
PROGNOSIS OF SURGERY IN ANGLE-CLOSURE GLAUCOMA

Operative Procedure	Number of Eyes	Postoperative Results (Number of Eyes)		
		Intraocular Pressure Controlled Without Medical Therapy	Intraocular Pressure Controlled With Medical Therapy	Intraocular Pressure Uncontrolled on Medical Therapy
Initial Surgery				
Peripheral Iridectomy	100	68 (68%)	18 (18%)	14 (14%)
Iridencleisis	82	41 (50%)	23 (28%)	18 (22%)
Total	182	109 (60%)	41 (23%)	32 (17%)
Secondary Iridencleisis	27	8 (30%)	8 (30%)	11 (40%)
Total operations	209	117 (56%)	49 (23%)	43 (21%)
Total eyes	182	117 (64%)	49 (27%)	19 (9%)

TABLE 3

RELATIONSHIP BETWEEN PREOPERATIVE TONOGRAPHY AND THE RESULTS OF INITIAL SURGERY IN ANGLE-CLOSURE GLAUCOMA

Preoperative Outflow Facility	Number of Eyes	Postoperative Results (Number of Eyes)		
		Intraocular Pressure Controlled Without Medical Therapy	Intraocular Pressure Controlled With Medical Therapy	Intraocular Pressure Uncontrolled on Medical Therapy
>0.20	60	51 (85%)	7 (12%)	2 (3%)
0.16–0.20	39	26 (67%)	8 (20%)	5 (13%)
0.11–0.15	36	18 (50%)	11 (31%)	7 (19%)
≤0.10	47	14 (30%)	15 (32%)	18 (38%)
Total	182	109 (60%)	41 (23%)	32 (17%)

failure rate in eyes with facility greater than 0.20 to a 38-percent failure rate in eyes with facility of 0.10 or less.

The relationship between initial outflow facility and prognosis comes into sharper focus when the preoperative outflow facilities are considered separately for each of the initial operative procedures. In Table 4 the results of peripheral iridectomy as an initial operation are compared with the preoperative outflow facility. With this procedure there are no failures among the 47 eyes with an initial outflow facility greater than 0.20 and only one failure among 71 eyes with an outflow facility greater than 0.15. However, 10 (77 percent) of the 13 eyes with an outflow facility of 0.10 or less failed to be controlled by peripheral iridectomy as an initial operative procedure. On the other hand, such impressive differences in prognosis as related to preoperative facility are not as apparent in those eyes where iridencleisis was performed as the initial operative procedure (table 5). The failure rate in this group remains very much the same at all outflow facilities. The sharp contrast between iridectomy and iridencleisis failure rates as a function of preoperative outflow facility is demonstrated graphically in Figure 1.

Of the 14 failures of peripheral iridectomy, 11 had an iridencleisis as a secondary operation. Only three (27 percent) of these failed to be controlled. It would appear that the results of iridencleisis in such eyes previously subjected to peripheral iridectomy were as successful as initial iridencleisis. On the other hand, 16 of the 21 iridencleisis failures were reoperated an half of these were again failures (table 6).

TABLE 4

PREOPERATIVE TONOGRAPHY AND THE RESULTS OF PERIPHERAL IRIDECTOMY AS AN INITIAL OPERATION IN ANGLE-CLOSURE GLAUCOMA

Preoperative Outflow Facility	Number of Eyes	Postoperative Results (Number of Eyes)		
		Intraocular Pressure Controlled Without Medical Therapy	Intraocular Pressure Controlled With Medical Therapy	Intraocular Pressure Uncontrolled on Medical Therapy
>0.20	47	42 (89%)	5 (11%)	0 (0%)
0.16–0.20	24	18 (75%)	5 (21%)	1 (4%)
0.11–0.15	16	8 (50%)	5 (31%)	3 (19%)
≤0.10	13	0 (0%)	3 (23%)	10 (77%)
Total	100	68 (68%)	18 (18%)	14 (14%)

TABLE 5

PREOPERATIVE TONOGRAPHY AND THE RESULTS OF IRIDENCLEISIS AS AN INITIAL OPERATION IN ANGLE-CLOSURE GLAUCOMA

Preoperative Outflow Facility	Number of Eyes	Postoperative Results (Number of Eyes)		
		Intraocular Pressure Controlled Without Medical Therapy	Intraocular Pressure Controlled With Medical Therapy	Intraocular Pressure Uncontrolled on Medical Therapy
>0.20	13	9 (70%)	2 (15%)	2 (15%)
0.16–0.20	15	8 (53%)	3 (20%)	4 (27%)
0.11–0.15	20	10 (50%)	6 (30%)	4 (20%)
≤0.10	34	14 (41%)	12 (35%)	8 (24%)
Total	82	41 (50%)	23 (28%)	18 (22%)

DISCUSSION

A. DIAGNOSIS

It is well recognized that many eyes with angle-closure glaucoma fail to respond with a significant pressure rise when subjected to a mydriasis provocative test. In early studies on tonography it became apparent that dilatation of the pupil either by darkroom or mydriatics could flatten the tonographic tracing in some eyes without inducing comparable elevations in intraocular pressure.[1] It was suggested that tonography might provide a more accurate means for the earlier recognition of the capacity for angle-closure in eyes with narrow angles. Interestingly enough, this hypothesis has been confirmed independently for the darkroom provocative test[2] as well as for the mydriasis provocative test.[3] Coincidentally, in both series the angle-closure group could be separated from other eyes by choosing almost identical values as a significant decrease in outflow facility (30 percent after darkroom mydriasis and over 25 percent after euphthalmine).

In this study a significant rise in intraocular pressure (8.0 mm.Hg or more) was found to occur in less than half of the normotensive eyes with clinical evidence of a capacity for angle-closure (table 1). This is in good agreement with results reported by others.[7] Leydhecker observed a similar rise in 46 percent of 57 such eyes after homatropine.[8] Foulds reported a rise of more than 8.0 mm.Hg in 17 of 41 normotensive angle-closure eyes following a darkroom test.[9] Furthermore, in this latter series as well as in the present study, the capacity for angle-closure could be recognized in 85 to 90 percent of the same eyes by carrying out tonography before and after the mydriasis. The mechanism by which a pressure rise

TABLE 6

RESULTS OF IRIDENCLEISIS AS A SECONDARY OPERATION AFTER FAILURES OF INITIAL SURGERY IN ANGLE-CLOSURE GLAUCOMA

Initial Operation	Number of Eyes	Postoperative Results of Secondary Surgery (Number of Eyes)		
		Intraocular Pressure Controlled Without Medical Therapy	Intraocular Pressure Controlled With Medical Therapy	Intraocular Pressure Uncontrolled on Medical Therapy
Iridectomy	11	5 (46%)	3 (27%)	3 (27%)
Iridencleisis	16	3 (19%)	5 (31%)	8 (50%)
Total Re-operations	27	8 (30%)	8 (30%)	11 (40%)

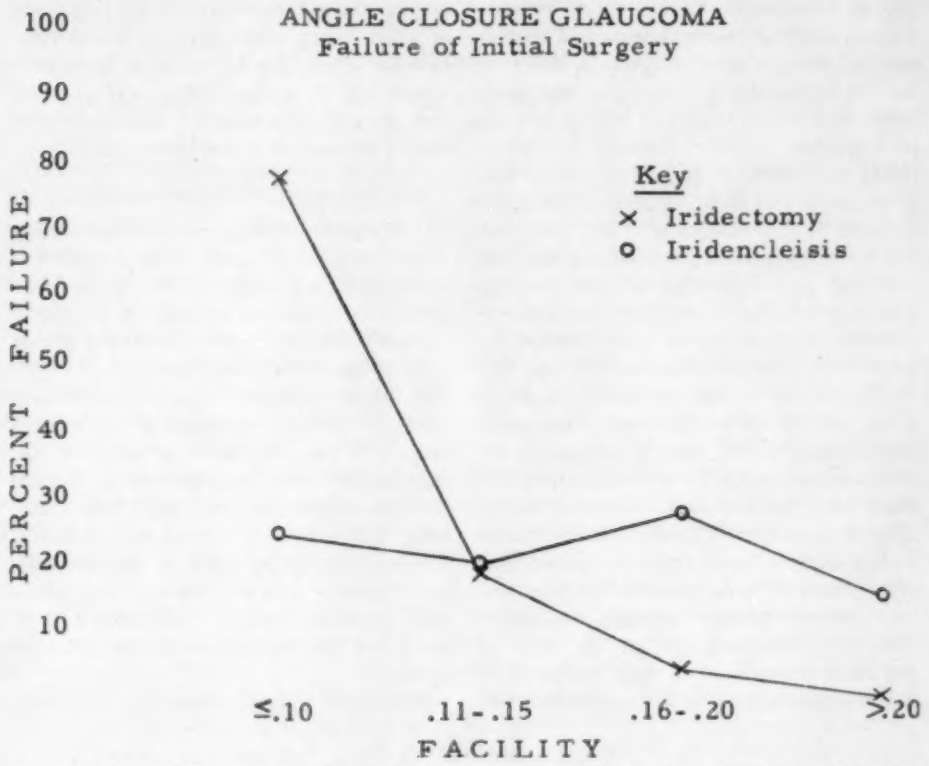

Fig. 1 (Becker and Thompson). The relationship between preoperative outflow facility (on miotic therapy) and the prognosis of initial surgery (iridectomy or iridencleisis).

is avoided when the outflow channels are partially occluded remains unexplained. At any rate it is apparent that all patients whose eyes present the gonioscopic picture of narrow angles of even moderate degree should be subjected to a mydriasis provocative test combined with tonography. Facility measurements permit earlier and more accurate diagnosis than do pressure measurements alone. By this means tonography as a supplement to the history and gonioscopic examination often simplifies the surgical therapy needed for a cure.

B. Therapy

Such rules of thumb as the history of previous attacks, the length of time in an attack, or even the extent of the peripheral anterior synechias provide only suggestive evidence of the need for a filtering operation in angle-closure glaucoma. These important clinical criteria cannot always answer the key question: Are the outflow channels adequate for normalizing pressure if closure can be avoided (by iridectomy), or has sufficient damage been done to necessitate a filtering procedure? Tonography offers an approach to the evaluation of patency of the outflow system which complements the gonioscopic findings.[4]

From the data collected in this series it is clear that a preoperative facility value greater than 0.15 (on miotic therapy) is adequate for pressure control in most eyes (fig. 1). In such eyes there is therefore no indication to do more than a simple peri-

pheral iridectomy. Under these circumstances, however, some patients will require medical therapy after surgery in order to control intraocular pressure. On the other hand, eyes with a facility of 0.10 or less are probably best subjected to iridencleisis as an initial procedure. A rare exception in this group is the eye in which outflow facility is reduced by iris contact with the trabecula, but without synechiae or trabecular damage. Some of these eyes may be recognized by gonioscopy after deepening the anterior chamber with saline, as recommended by Shaffer.[10] At facilities between 0.10 and 0.15 the failure rate is much the same for iridectomy and iridencleisis. It is our current practice to subject such eyes to peripheral iridectomy and to reserve iridencleisis for those eyes that fail to be controlled by the simpler procedure. The trend toward more iridectomies is based upon the greatly decreased risk of a flat anterior chamber and its resultant further damage to outflow channels. Iridectomy also avoids some of the other hazards which may follow filtering procedures, such as late endophthalmitis, pressure rises associated with scarring down of a previously functioning bleb, and cataract formation. Furthermore, an iridectomy which fails to control intraocular pressure does not appear to alter the chances for successful subsequent iridencleisis.

Summary and Conclusions

1. A significant decrease in outflow facility (more than 25 percent) after a mydriasis provocative test affords one of the best means of recognizing the capacity for angle-closure glaucoma in eyes with narrow angles.

2. Preoperative tonography in eyes with angle-closure provides useful complementary data for the decision as to the type of surgery to be performed. An adequate outflow facility (over 0.15) assures the surgeon that the simpler and safer peripheral iridectomy will suffice to control the glaucoma. Filtering operations, such as the iridencleisis, should be reserved for eyes with markedly impaired outflow facilities (0.10 or less) and for the failures of initial iridectomies.

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PATHOLOGY OF THE ANGLE OF THE ANTERIOR CHAMBER IN PRIMARY GLAUCOMA*

A PRELIMINARY REPORT

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Continued and increasing interest in the angle of the anterior chamber from clinical, histologic, physiologic, and pathologic points of view is manifested by the numerous papers on the subject in recent ophthalmic literature.[19-24] Many of these investigators examined the angle and especially the trabeculae from different viewpoints with special staining and techniques to learn its intimate histology. All have contributed to a better understanding of the subject. Of basic importance is the work of Theobald[19] whose serial sections of the eye and the models made therefrom have established many facts before considered controversial. The most recent interesting finding was the demonstration by Zimmerman[24] of the presence of an hyaluronidase-sensitive acid mucopolysaccharide in the trabeculae and iris in adult human eyes. This subject is important because of the light it may throw on the problem of primary glaucoma and specifically on the causes for the obstruction to the outflow of aqueous which is believed to be the fundamental problem.

Over the past 12 years, it has been possible for us to obtain the eyes of aged persons for pathologic study. The primary objective was to determine the changes normally taking place in such old eyes untroubled by other ocular pathology. During this period it has been our good fortune to get eyes which have been affected by chronic simple glaucoma and acute episodes of congestive glaucoma but which have not had any surgical intervention. We now have accumulated 11 such eyes from seven patients. In this paper, the changes in the angle of the anterior chamber will be stressed.

This paper is considered to be a preliminary report. Work is now in progress on several additional glaucomatous eyes that are being studied by serial section and with four different staining techniques following the method described by Teng et al.[9] Such a study will enable us to examine the entire angle which we were unable to do with our initial group of eyes.

The material for this study was obtained at The Home for Aged and Infirm Hebrews of New York City. The residents of the home range in age from 65 years up to 90 years and over. There is a complete medical history of each resident which is maintained from the time of admission up to the time of death. Pertinent laboratory data and X-ray studies were done whenever necessary. In a large percentage of cases, up to 85 percent, complete post-mortem examinations were made, including the eyes and the brain. In this way a large amount of material was made available for study. Over 300 post-mortem eyes have been examined for the changes commonly present in old age and for special studies of retinal circulation and the macula. It is from this reservoir of material that the eyes with primary glaucoma were selected for detailed study.

Not too much is known about the pathology in the drainage system centering around the angle of the anterior chamber in primary glaucoma. Most of the reports have been based on studies of pathologic material either postoperative or in absolute glaucoma where,

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as a result of prolonged increased pressure, widespread degenerative changes and atrophic conditions have resulted. The changes that were seen in three eyes with chronic simple glaucoma, as reported by Duke-Elder,[1] were minimal. In one the angle was free, and in the other two it was infiltrated to some extent with cells and pigment.

Gonioscopy has been a great help in estimating the clinical pathology of the chamber angle, and numerous reports beginning with Salzmann in 1915,[2] and Troncoso, in 1925,[3] attest to the value of this type of observation for clinical evaluation of the glaucomatous patient. Valuable information about the rate of outflow of the aqueous in normal and glaucomatous eyes has resulted from the tonographic studies developed by Morton Grant.[4] The work on aqueous veins by Ascher[5] and Goldmann[6] has also contributed to our knowledge of the physiology of aqueous flow in normal and pathologic eyes. But fundamental studies on the causes of obstruction to the normal outflow of aqueous in early glaucomatous eyes is still lacking.

Recent exhaustive and painstaking work on the anatomy and histology of the angle of the anterior chamber and Schlemm's canal has contributed much our understanding of these structures. Georgiana Theobald's[7] work on reconstruction of the Schlemm canal and the collecting tubules from serial sections gave a three dimensional view of this region. Ashton[8] enlarged on this by his technique of injection of Neoprene, thus making a plastic model of Schlemm's canal and its tributaries and demonstrating aqueous veins.

In 1954, Teng, Paton, and Katzin[9] demonstrated at an exhibit at the annual convention of the American Academy of Ophthalmology and Otolaryngology three glaucomatous eyes examined by serial section. The exhibit showed almost the entire angle of the anterior chamber, and Schlemm's canal. In many areas the angle was obliterated by anterior synechia. In other places, the canal of Schlemm was partially closed by degenerative changes and by proliferative endothelial tissue. All these pathologic changes may contribute to the etiology of glaucoma by damming back the outflow and increasing the intraocular pressure.

Our patients are all old persons and perhaps not perfectly representative of the typical early glaucoma found in middle-aged adults in whom chronic simple glaucoma most frequently begins. But they are eyes that have had increased tension for prolonged periods, usually controlled by medical means and have never been subjected to eye surgery up to the time of death.

Case reports

Case 1

S. M., a white woman, aged 74 years at the time of death, was first discovered to have glaucoma on November 25, 1942. The tension was 38 mm. Hg in the right eye and 33 mg. Hg in the left eye (Schiøtz). The vision was: O.D., 20/40; O.S., 20/30. The angle clinically was of the wide-open type. The fields of vision were normal. Tension was controlled by pilocarpine (two percent, four times daily) in each eye. When last examined on February 3, 1943, two and one-half months later, the tension was 28 mm. Hg in each eye. She died on February 28, 1943. The general diagnoses at the time of death were generalized arteriosclerosis, hypertension, coronary artery thrombosis, myocardial infarction, and arteriosclerotic closure of peripheral vessels.

Both eyes were obtained post mortem and fixed in Bouin's solution and embedded in celloidin. They were then sectioned and stained with hematoxylin and eosin. Other stains were not done at this time. Representative sections were photographed under low, medium, and high-power magnifications.

The angles are all open on these slides. The trabeculae of the anterior chamber show some thickening and sclerosis. The interesting findings are in the appearance of Schlemm's canal. On some sections the canal appears as a thin slit. On other sections it appears as two small openings separated by epithelial cells and hyaline-like tissue. Considerable pigment cells and granules are present amongst the trabeculae. The trabeculae appear to be crowded together and to have lost the appearance of regular individual strands (fig. 1).

Case 2

A. M., a white woman, aged 90 years at the time of death, was first seen in the eye clinic on August 31, 1938. Her vision at that time was O.D., 20/30; O.S., 20/40. Ocular tension was normal. On De-

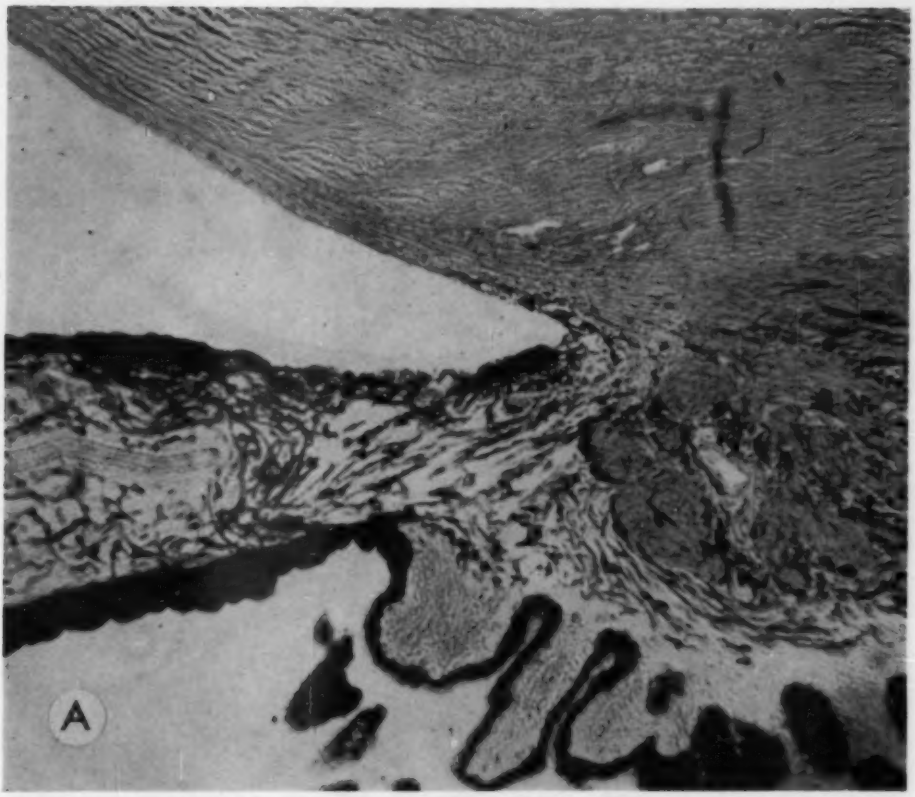

Fig. 1A (Kornzweig, et al.). Medium-power section through the angle of anterior chamber, showing open angle, thickened compressed trabeculae, narrowed and partially obliterated Schlemm's canal, and collecting tubules.

cember 20, 1940, she had an attack of acute congestive glaucoma in the right eye which was controlled by the use of miotics. When seen again in the clinic on August 13, 1941, her vision was 20/100 in the right eye and 20/70 in the left. Ocular tension was 23 mm. Hg (Schiøtz) and it was controlled by pilocarpine (two percent, three times a day). The anterior chamber was shallow. Gonioscopy study was not done. The fields of vision as far as could be determined in this patient were fairly normal.

She died on March 22, 1943, with the general diagnoses of diabetes mellitus, hypertension and generalized arteriosclerosis, and chronic congestive glaucoma. The eyes were obtained at post mortem and fixed in Bouin's solution and embedded with the usual technique in celloidin. Numerous sections of the anterior segment were made and studied.

In this series of slides several sections show an open angle, others a partially occluded angle, and still others a completely occluded angle by peripheral anterior synechias. The trabeculae nevertheless show a fairly normal configuration and Schlemm's canal and such collecting tubules as are seen appear to be of normal caliber and patency. Pigment granules are minimal in amount, which is somewhat surprising in view of the patient's diabetes mellitus (fig. 2).

CASE 3

M. M., a white woman aged 81 years at the time of death, was first seen in the eye clinic on August 18, 1943. The vision was: O.D., 20/40; O.S., 20/50. The tension in the right eye was 24 mm. Hg (Schiøtz); in the left eye the tension was too soft to register on the Schiøtz tonometer. This was due to a trephination on the left eye done in 1932. She had been using pilocarpine (two percent) in the right eye since the operation. The right eye had a deep anterior chamber. The left eye showed a

Fig. 2A.* Open angle.

* Figs. 2A, 2B and 2C (Kornzweig, et al.). Medium-power view of three sections of the angle of the anterior chamber. The trabeculae show a fairly normal configuration. Schlemm's canal and the collecting tubules appear to be of normal caliber and patency.

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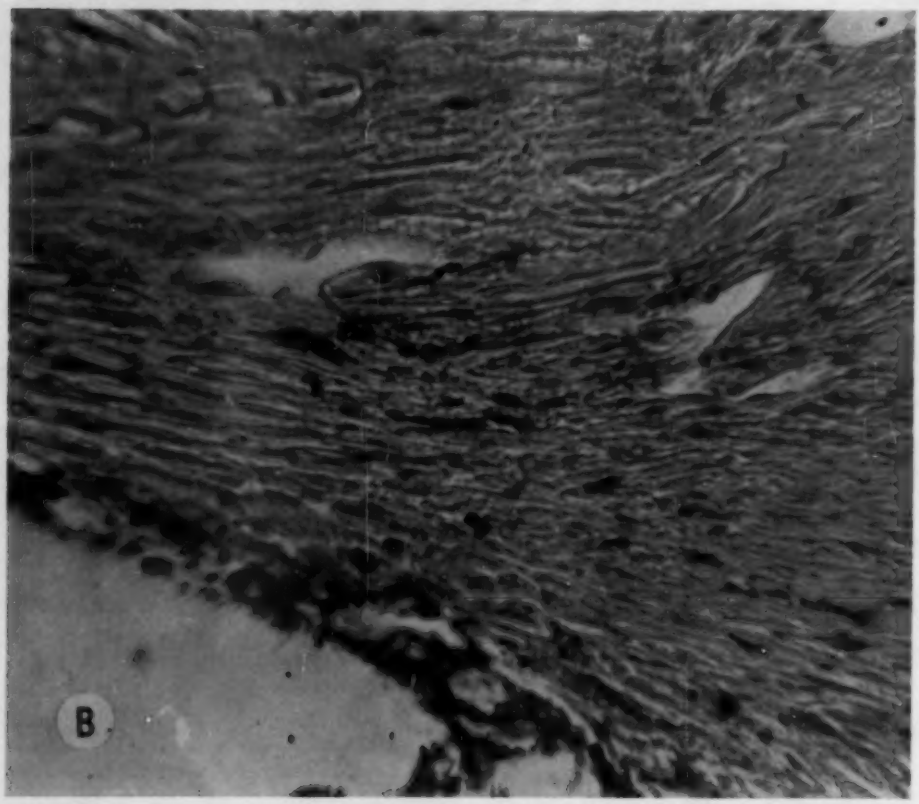


Fig. 1B (Kornzweig, et al.). High-power view of trabeculae of Figure 1A. The trabeculae are thickened and compressed. The intertrabecular spaces are almost completely obliterated. Pigment granules and cells are present among the strands. Schlemm's canal is partially obliterated by hyalinelike tissue and epithelial cell proliferation.

large conjunctival bleb and peripheral iridectomy. The fields of vision were normal. The fundi could be seen and showed slight cupping of the optic discs. She was observed at regular intervals until March 8, 1944. At no time did the ocular tension in the right eye go above 26 mm. Hg. In the last two visits to the clinic, the tension in the right eye fell to 17 mm. Hg and pilocarpine was discontinued.

She died on April 4, 1944. The final diagnoses were hypertension, diabetes mellitus, lymphosarcoma, and chronic simple glaucoma.

The eyes were obtained in the usual manner, and prepared for section and microscopic examination. The sections of the right eye, which had never been operated on, were studied for changes in the angle of the anterior chamber. The angles are open, and the trabeculae appear somewhat thickened and

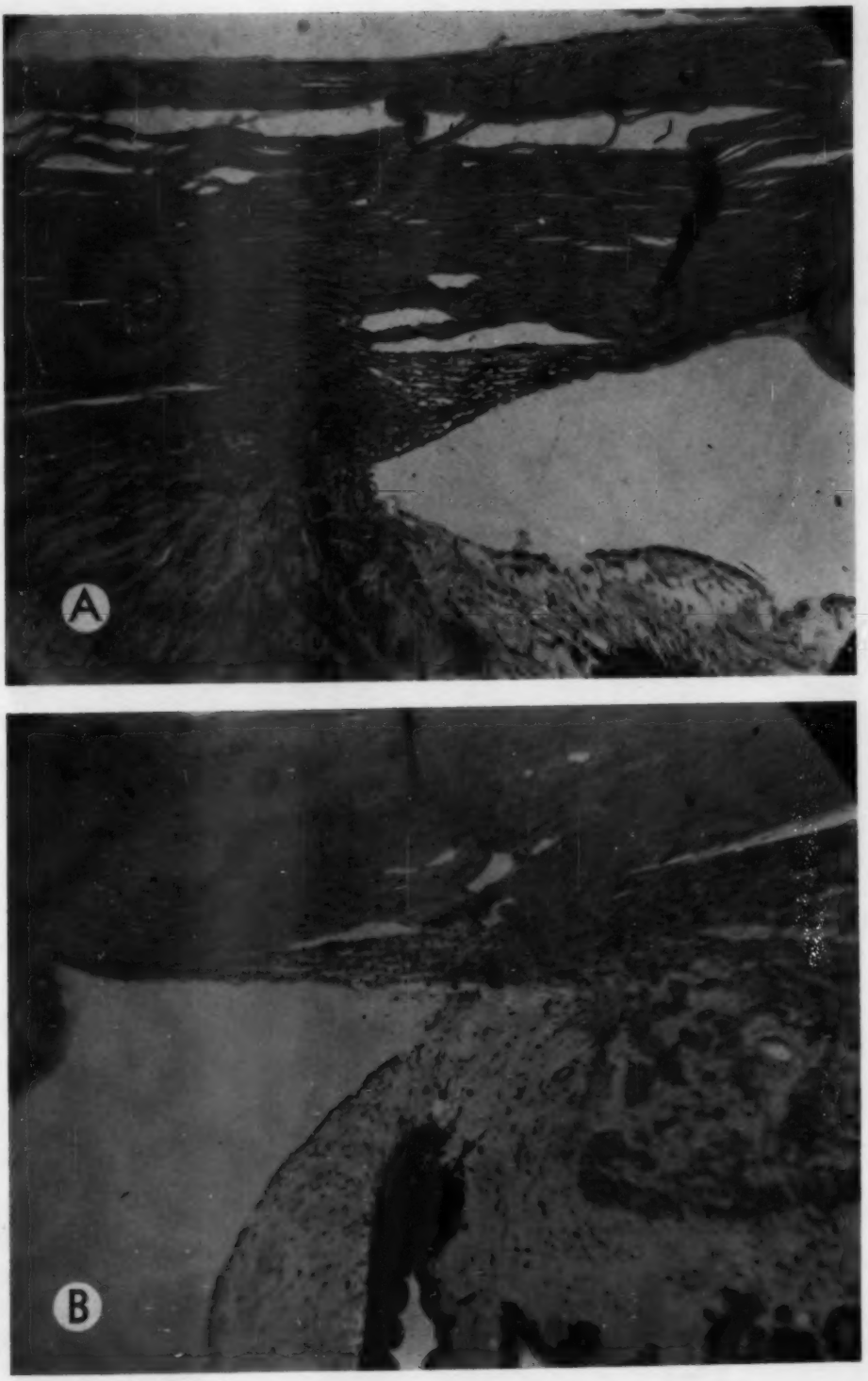


Fig. 2B. Partially occluded angle.

Fig. 3A (Kornzweig, et al.). Medium-power view of section through the angle of the anterior chamber. The trabeculae have a fairly normal configuration. Schlemm's canal and the collecting tubules are normal and patent.

Fig. 2C. Completely occluded angle.

sclerosed. The intertrabecular spaces are narrowed and almost obliterated. Schlemm's canal appears to be normally patent and to have a regular epithelial lining.

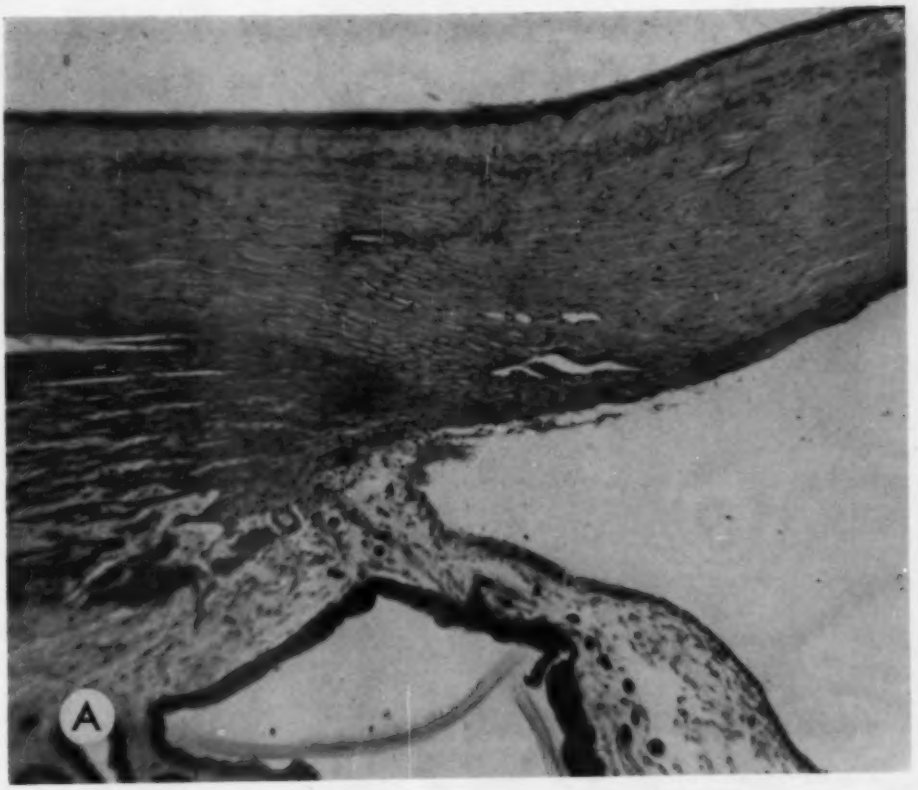
In front of the trabeculae, on several slides, there is a pigmented tissue which looks like it might be debris, or possibly trabeculae that were separated from the main body of the trabecular tissue. Similar looking debris is noted on the posterior surface of the cornea. It does not look like fuzz noted by Theobald[20] in pseudo-exfoliation of the lens capsule, a possible cause of secondary glaucoma. There is little evidence of cause for obstruction to aqueous outflow in this eye (fig. 3).

CASE 4

J. C., a white man, aged 80 years at the time of death, was first seen in the eye clinic on March 4, 1948. At that time vision was: O.D., 15/40; O.S., 15/25. The anterior chamber was of normal depth, the lens showed incipient cataractous changes, and the fundi were relatively normal aside from narrowed arteries with alteration in caliber. There was no cupping of the disc.

When seen a year later, the vision had dropped to 15/70 in the right eye, and 15/50 in the left eye. There was in addition, a rise in intraocular pressure to 38 mm. Hg (Schiøtz) in the right eye and 24 in the left. Two weeks later the tension was

Fig. 3B (Kornzweig, et al.). High-power view of section in (A). The trabeculae are normal in appearance but somewhat thickened and sclerosed. The intertrabecular spaces are narrowed. The debris in front of the trabeculae is of undetermined origin, possibly trabecula separated from the main body.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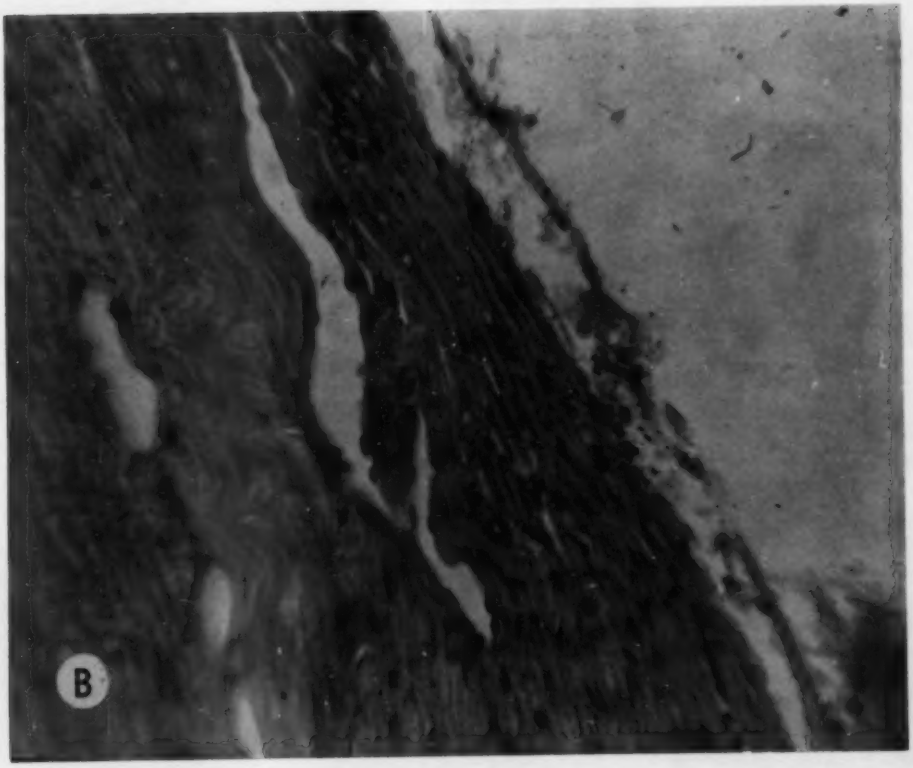
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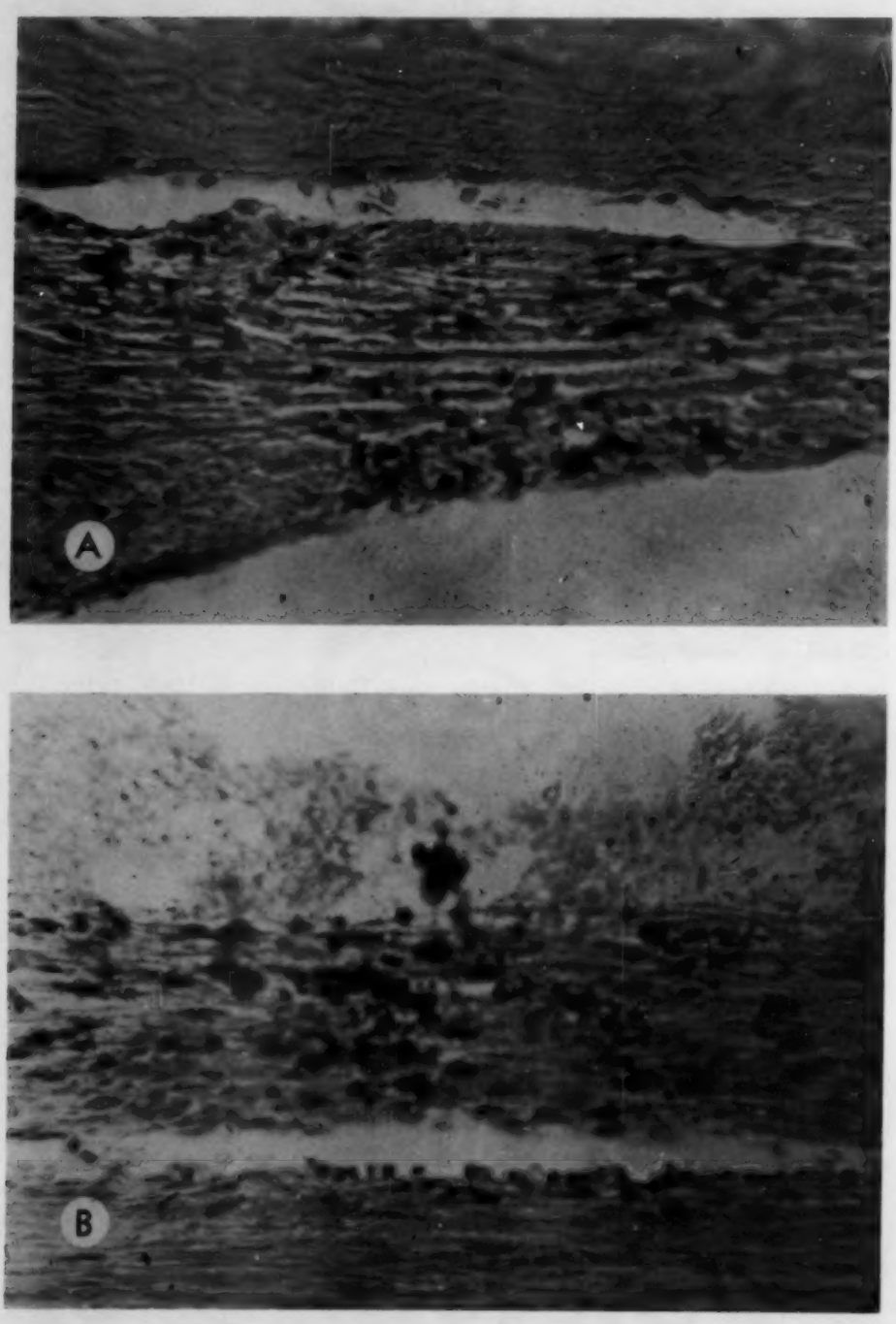
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Fig. 4A (Kornzweig, et al.). High-power view of a section of the angle of the anterior chamber. The trabeculae are thickened and sclerosed. The intertrabecular spaces are narrowed and obliterated in places. Schlemm's canal is patent but there is some evidence of proliferation of endothelial cells.

43 mm. Hg, O.D., and 28 mm. Hg, O.S. A recheck the following day showed a tension of 35 mm. Hg in each eye. A diagnosis of chronic simple glaucoma was made and the patient placed on pilocarpine.

The tension was controlled for awhile, but when seen on May 2, 1949, the tension had risen to 43 mm. Hg in the right eye and 38 mm. Hg in the left. Eserine salicylate (0.5 percent) was then added to the pilocarpine, for a short period, and by May 10, 1949, the tension was reduced to 24 mm. Hg in the right eye and 20 mm. Hg in the left. The tension was controlled finally by using pilocarpine (four percent) five times a day.

The patient was last seen in the eye clinic on June 13, 1950, at which time his mental condition and lack of co-operation prevented further tonometric studies. The optic discs showed some cupping and beginning atrophy. The patient was continued on three-percent pilocarpine, five times a day, and one drop of eserine (0.5 percent, twice a day) up to the date of death, March 26, 1951. The diagnoses at that time were hypertension, pulmonary emphysema, generalized arteriosclerosis, cerebral concussion, incipient cataracts, and chronic simple glaucoma.

Numerous sections through the anterior chamber angle were made of both eyes. The angles were open on all slides. The trabeculae on most sections showed a fairly normal histologic appearance but the collagenous core appeared thicker than normal, so that the trabeculae were crowded together in many places. The canal of Schlemm was patent and open on most slides. Several sections taken to one side of the eye showed a long stretch of the canal. On this slide, the canal was narrowed and in one or two places obliterated. Pigment cells and granules were moderate in amount, and did not appear to be important.

This case of typical chronic simple glaucoma showed very little in the angle studies to account for the clinical picture. The only positive findings were thickening of the trabeculae and several areas of narrowing and occlusion of Schlemm's canal (fig. 4).

Case 5

L. H., a white man, aged 83 years at the time of death, was first seen in the eye clinic on June 22, 1949. At that time his vision was reduced to finger counting at two feet in the right eye and light perception in the left eye. The tension was 43 mm. Hg (Schiøtz) in each eye despite the fact that he was using pilocarpine (two percent) three times a day. The fields of vision were restricted to within five degrees of fixation. The pupillae were small but the optic discs could be seen and appeared atrophic and cupped. The anterior chamber was of normal depth. Gonioscopic examination was not performed because of a small corneal ulcer in the left eye. The pilocarpine was increased to three percent, five times a day. The patient was last seen on August 29, 1950, at which time the tension was 40 mm. Hg in each eye. He died on June 6, 1951, and the diagnoses were hypertension, pulmonary emphysema, diabetes mellitus, status after suprapubic prostatectomy, and chronic simple glaucoma.

Both eyes were obtained in the usual manner, fixed, embedded in celloidin, and sectioned. The angles of the anterior chamber were open except for slight encroachment of the root of the iris, not sufficient, however, to cover the trabeculae. The trabeculae showed considerable thickening of the collagenous core, with crowding together and obliteration of the intertrabecular spaces.

Most of the pathology appeared to be in Schlemm's canal. Almost all sections showed marked narrowing and obliteration of Schlemm's canal chiefly by proliferation of endothelial tissue. Pigment cells and granules were scattered throughout the trabeculae. This picture was seen in both eyes and in most sections studied. This would indicate that there existed considerable interference with the outflow of aqueous in these eyes. Several sctions also showed a washed out appearance, stressed by Teng and associates believed to be indicative of degeneration (fig. 5).

Case 6

R. L., a white woman, aged 96 years at time of death, was seen in the eye clinic on August 27, 1947. Her vision was 15/40 in the right eye and 15/70 in the left eye, with correction. The fundi showed moderate cupping and grayness of the optic discs. The ocular tension was 23 mm. Hg in each eye (Schiøtz). The angles of the anterior chamber were of moderate depth. Gonioscopic examination

Fig. 4B (Kornzweig, et al.). Sagittal section of the angle showing a long stretch of Schlemm's canal. The proliferation of endothelial cells and partial obliteration of the canal at one spot are seen.

Fig. 5A (Kornzweig, et al.). Medium-power view of a section through the angle of the anterior chamber, showing slight encroachment of the root of the iris.

and peripheral fields were difficult to do because of poor co-operation.

Over a period of months, the patient's vision diminished to hand movements in spite of the use of pilocarpine (two percent) several times a day. Part of this failing vision was due to senile macular degeneration and nuclear lens sclerosis. The tension never went above 23 mm. Hg.

At the time of death, the diagnoses listed were hypertension, arteriosclerotic heart disease, myocardial insufficiency, senile macular degeneration, and chronic simple glaucoma of the low tension type.

The examination of the eye sections showed open angles, thickening and crowding of the trabeculae, and obliteration of the intertrabecular spaces. Schlemm's canal was present but narrowed and in places obliterated. Pigment cells were present in moderate numbers. These slides would indicate the possibility of obstruction to aqueous outflow in the appearance of the trabeculae and the narrowing of Schlemm's canal (fig. 6).

CASE 7

K. M., a white woman, aged 71 years at the time of death, is the last case. She was at the home for only a short period, from June 6, 1954, to July 12, 1954. At the time of admission, it was known that she had chronic simple glaucoma which had progressed to absolute glaucoma in the right eye. This eye had had a posterior sclerotomy before admission in a futile attempt to control the progress of the disease in this eye. There was no light perception in this eye and the tension varied from 60 to 90 mm. Hg. The vision in the left eye was 20/70 with correction. The anterior chamber was of moderate depth, the pupil was pinpoint in size. The fundus could not be seen. The field of vision in the left eye showed a typical contraction in the lower nasal quadrant and an enlargement of the blindspot. The

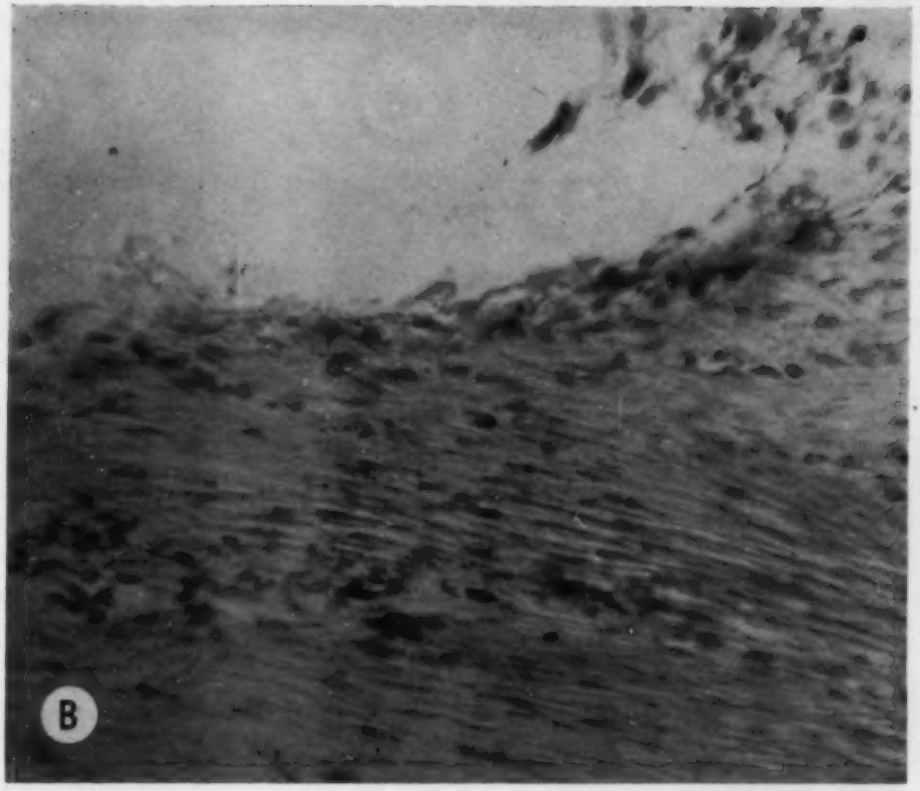

Fig. 5B (Kornzweig, et al.). High-power view of the trabecula, showing thickening and obliteration of the intertrabecular spaces. Schlemm's canal and the collecting tubules are almost completely obliterated by endothelial proliferation and compression.

tension in the eye was 23 mm. Hg with the use of pilocarpine (two percent, four times a day). The patient died shortly after admission of cerebral thrombosis.

Both eyes were obtained but this report will be limited to the left unoperated eye.

The angle of the anterior chamber was open on all sections. The trabeculae were fairly well defined and showed only slight to moderate thickening of the collagenous core. The intertrabecular spaces are open but filled with pigment cells and granules. Schlemm's canal wherever it showed appeared to be of normal patency and normal epithelial lining. Collecting tubules wherever they showed were open.

Differential staining with Masson's trichronic stain and Van Giesen's elastic tissue stain added clarity to the slides but little additional information to the pathology. One got the impression from the elastic tissue stain that there was a diminution in elastic fibrils on the trabeculae but these are usually very difficult to demonstrate. An occasional slide showed a partial obliteration of Schlemm's canal (fig. 7).

Control case

For the sake of comparison, the eye of a 78-year-old woman who did not have glaucoma is shown. The angle is cove-shaped and well defined. The trabeculae stand out individually, and the intertrabecular spaces are open. Pigment granules are present, though in small amounts. Schlemm's canal is patent throughout and wide open, with a single layer of endothelial cells. Collecting tubules where seen are open endothelial-lined ducts (fig. 8).

Comments

Obstruction to the outflow of the aqueous from the anterior chamber has been postulated as a cause for primary glaucoma of the

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Fig. 6A (Kornzweig, et al.). Medium-power view of a section through the angle of the anterior chamber, showing an open angle, but narrowing and partial obliteration of Schlemm's canal.

Fig. 7A (Kornzweig, et al.). Medium-power view of a section through the angle of the anterior chamber. The angle is open. Schlemm's canal and collecting tubules are patent and fairly normal in appearance.

Fig. 6B (Kornzweig, et al.). High-power view of the trabecula, showing thickening and crowding together of the trabeculae, resulting in narrowing of the intertrabecular spaces. Schlemm's canal seen in the lower corner is partially obliterated.

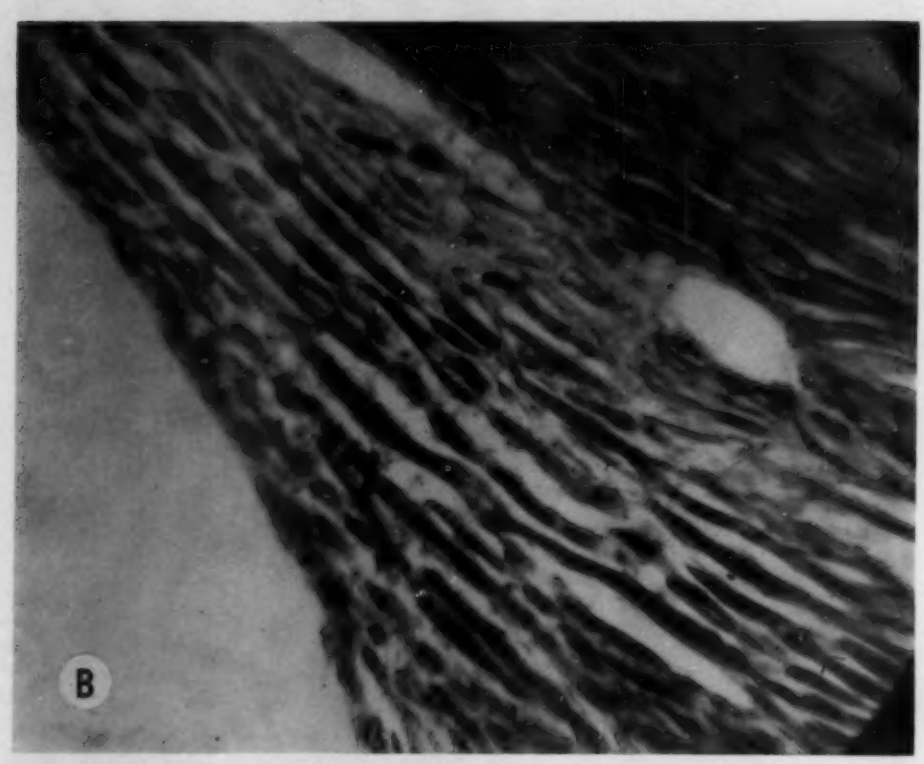

Fig. 7B (Kornzweig, et al.). High-power view of Figure 7A. Some narrowing and obliteration in one spot is noted of Schlemm's canal. The trabeculae appear normal except toward the outer fourth where they are paler staining and have a washed-out appearance, suggesting primary degeneration.

wide-angle type, rather than hyperformation of aqueous by the ciliary process. Friedenwald[11] hypothesized that the reabsorption of the aqueous from the anterior chamber into Schlemm's canal is conditioned by the presence of small amounts of plasma in the canal which attracts the aqueous by its osmotic tension. This hypothesis presupposes a communication between Schlemm's canal and the anterior scleral plexus of blood vessels. In primary glaucoma, this plasma is present in insufficient quantities due perhaps to sclerosis of the anterior scleral plexus of vessels. This theory was corroborated to some extent by the work of Kronfeld[12] and his associates on the blood-filling phenomenon of Schlemm's canal. The eye is decompressed by withdrawing aqueous from the anterior chamber with a hypodermic needle or compressed with Kukan's ophthalmodynamometer. In normal eyes the presence of blood in Schlemm's canal could be readily seen after decompression. In primary glaucoma with wide angles, the blood either failed to appear in the Schlemm's canal after decompression or else appeared as a thin band, a segmented band, or in isolated spots, with the rest of trabeculae opaque.

Further evidence for a diminution of the outflow of aqueous in primary wide angle glaucoma was noted by Moses and Bruno,[13] Goldmann,[14] and Morton Grant[15] with tonographic studies which showed a lower coefficient of outflow than in normal eyes. Ascher[16] saw fewer aqueous veins in wide-angle glaucoma, and noted a diminished and

slower flow of aqueous. Barkan[17] came to the same conclusion from his clinical studies and predicted that the obstruction to outflow of the aqueous would be found in the trabeculae or Schlemm's canal.

In all of our eyes but one, the angles of the anterior chamber were open. In the one case with peripheral anterior synechias, there was a history of repeated attacks of acute congestive glaucoma. Even in this one case, several sections showed open angles and other sections only partially occluded angles. Several eyes showed encroachment of the angle by the stroma of the iris but no occlusion. The trabeculae of all the sections showed varying degrees of thickening and sclerosis with corresponding narrowing and obliteration of the intertrabecular spaces. Pigment granules and cells were present to some extent in all eyes scattered throughout the trabeculae. Schlemm's canal showed various degrees of patency from normal to almost complete occlusion. When it was occluded, the cause seemed to be endothelial proliferation or compression by thickened or degenerated trabeculae. All of these factors, by narrowing or occluding the outflow channels for the aqueous from the anterior chamber, may help to raise the intraocular pressure.

Fig. 8A (Kornzweig, et al.). Medium-power view of a section of the angle of the anterior chamber of a normal aged eye without glaucoma. The angle is open and cove-shaped.

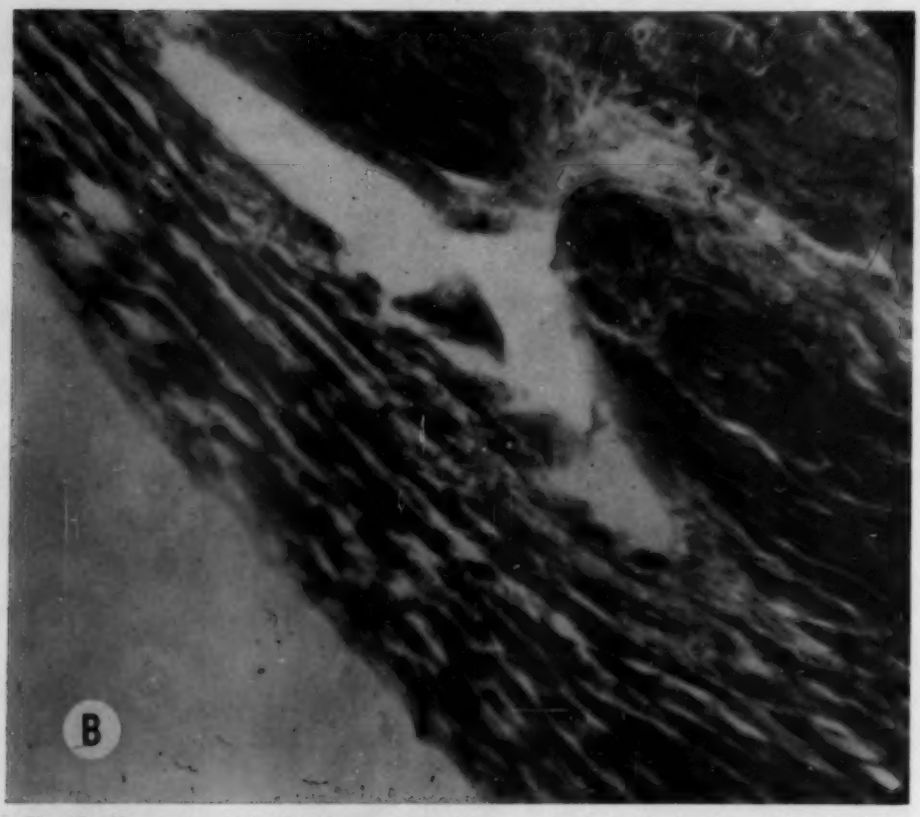

Fig. 8B (Kornzweig, et al.). High-power view of Figure 8A. The trabeculae show slight to moderate sclerosis. The intertrabecular spaces are open. Schlemm's canal and a small collecting tubule are patent and of normal caliber.

While our study of these eyes seems to add further evidence for the belief that the intraocular pressure is increased because of obstruction to outflow of the aqueous, it does not account for the cause of the changes noted in Schlemm's canal or in the trabeculae. Some of these changes, such as thickening of the trabeculae, have been found in eyes without glaucoma, though not so consistently.

Occlusion of Schlemm's canal, however, is rarely found in normal aged eyes without glaucoma. Pigment granules and cells have been seen in varying amounts in the intertrabecular spaces of almost all aged eyes. Perhaps these changes are a result of rather than a cause of increased intraocular pressure? Perhaps there is a chemical or functional cause for the increased intraocular pressure, such as is indicated by the work of Bloomfield[10] who found diminished acetylcholine in the aqueous of glaucomatous eyes after light stimulation, or the theory of Friedenwald[11] on the diminution of plasma in the canal of Schlemm. There is still much more work to be done to clarify this subject.

Summary

Eleven eyes from seven patients with primary glaucoma were examined for changes

in the angle of the anterior chamber. All of these eyes were obtained at post mortem at the Home and Hospital for Aged and Infirm Hebrews of New York City. All were treated medically while the patients were residents of the home and none had had any surgical intervention. The positive pathologic findings in the angle were:

1. Thickening and sclerosis of the trabeculae.
2. Narrowing or obliteration of the intertrabecular spaces.
3. Variation in the patency of the canal of Schlemm from narrowing and partial occlusion to complete occlusion.
4. Endothelial proliferation causing occlusion of Schlemm's canal.
5. Pigment granules and cells in varying amounts in the intratrabecular spaces.

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THE INFLUENCE OF SYSTEMIC STEROID THERAPY ON THE INTRAOCULAR PRESSURE*

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A considerable amount of data has been reported concerning the effects of local and systemic steroid therapy in the treatment of eye diseases; the indications and contraindications for the use of such drugs are well known to the ophthalmologic profession. On the other hand, information is limited as to the true effect on the intraocular pressure of these drugs (such as cortisone, hydrocortisone, and prednisone) when administered systemically in their usual fashion. Several published reports have suggested but not proved that cortisone influences intraocular pressure.[1] Patients with glaucoma may occasionally require cortisone or other steroid compounds for the treatment of ophthalmic or systemic diseases. Cortisone has been said in occasional cases to increase the intraocular pressure of primary open-angle glaucoma patients when administered systemically.[2] On the other hand, it is common experience that steroids may be applied locally without apparent adverse effect on the pressure. To provide additional data, in this paper are presented evaluations of the intraocular pressure, facility of outflow, and gonioscopy in a group of 13 patients with severe asthma who were under steroid therapy for a long period of time, but otherwise were apparently normal.

METHOD

The 13 patients with severe asthma were invited from the Allergy Clinic of the Massachusetts General Hospital for tonographic and gonioscopic studies. All of these patients had no ocular complaints or history of eye disease other than refractive errors. Nine of these patients had received cortisone, hydrocortisone, and prednisone. Of the remaining four patients, two were under cortisone therapy and two under prednisone therapy. Following a routine eye examination, tonography was carried out according to the method of Grant,[3] and gonioscopic examination was performed after tonography. All measurements of intraocular pressure in this investigation were made with the electronic Schiøtz tonometer (V. Mueller Co) and were expressed according to the 1948 calibration of the Committee on Standardization of Tonometers. All tonographic tracings were made on a Sanborn recorder. Facility of aqueous outflow (C) was calculated from the 1954 calibration of the Committee on Standardization of Tonometers; in calculating aqueous flow (F) the pressure in recipient veins was considered to be 10 mm. Hg.

FINDINGS

No ocular pathology was found on routine eye examination in these 13 severe asthmatic patients. The tonographic and gonioscopic findings indicated that in most instances there was no significant abnormality of intraocular pressure and facility of outflow in patients under prolonged steroid therapy (table 1). However, in Case 7 there were suggestive signs of open-angle glaucoma; in Case 2 the angle was found to be hazardously narrow, but outflow was normal. The one case (7) of probable open-angle glaucoma could well represent an incidental finding among persons over 40 years of age since no trend of glaucoma was discovered in the group as a whole. The average intraocular pressure was 20.35 mm.

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TABLE 1

TONOGRAPHIC AND GONIOSCOPIC FINDINGS ON PATIENTS WITH SEVERE ASTHMA WHO ARE UNDER A LONG PERIOD OF SYSTEMIC STEROID THERAPY

No. of Case	Name	Age (yr.)	Sex	Systemic Steroid Therapy			Gonioscopic Findings	Tonographic Findings		
				Medication	Period of Therapy	Av. Dose Daily (mg.)		I.O.P.	C.	F.
1	D.M.	63	M	Cortisone Hydrocortisone Prednisone	5/10/51–11/ 1/53 11/ 1/53– 5/ 9/55 5/ 9/55– 9/ 5/57	75 60 10	Wide and open with no exceptions	O.D. 14.5 O.S. 17	0.31 0.27	0.74 1.30
2	C.E.	58	M	Hydrocortisone	3/13/54– 9/ 5/57	50	Suspected subacute angle-closure glaucoma O.U.	O.D. 14.5 O.S. 13.5	0.25 0.23	0.60 0.39
3	E.R.	62	F	Prednisone*	4/16/55– 4/25/56	45	Wide and open with no exceptions	O.D. 18 O.S. 17	0.17 0.12	1.04 0.58
4	M.D.	60	F	Cortisone Hydrocortisone Prednisone	5/22/51–10/23/53 10/23/53– 4/ 8/55 4/ 8/55– 9/ 5/57	50 40 10	Wide and open with no exceptions	O.D. 18 O.S. 17	0.23 0.23	1.27 1.10
5	E.L.	40	M	Cortisone Hydrocortisone Prednisone	9/10/52– 5/ 2/54 5/ 3/54–10/13/55 10/13/55– 9/ 5/57	100 90 20	Wide and open with no exceptions	O.D. 25 O.S. 23.5	0.19 0.21	2.19 2.25
6	C.R.	65	M	Cortisone Hydrocortisone Prednisone	2/ 7/52–10/20/53 10/20/53– 5/ 7/55 5/ 8/55– 9/ 5/57	125 100 40	Wide and open with no exceptions	O.D. 25.5 O.S. 22	0.22 0.18	2.75 1.73

* Steroid therapy was discontinued, temporarily, at the time of examination.

(*continued on page 330*)

TABLE 1 (*continued*)

No. of Case	Name	Age (yr.)	Sex	Systemic Steroid Therapy			Gonioscopic Findings	Tonographic Findings		
				Medication	Period of Therapy	Av. Dose Daily (mg.)		I.O.P.	C.	F.
7	P.P.	53	M	Hydrocortisone Prednisone	9/25/54–11/19/55 11/19/55– 9/ 9/57	100 15	Wide and open with no exceptions	O.D. 19.5 O.S. 26.5	0.10 0.09	0.67 1.22
8	M.P.	64	F	Hydrocortisone	5/ 4/54– 5/ 1/56	100	Wide and open with no exceptions	O.D. 21 O.S. 21	0.23 0.21	1.91 1.74
9	N.Z.	56	F	Cortisone Prednisone	4/25/51– 4/28/57 4/29/57– 9/11/57	50 15	Wide and open with no exceptions	O.D. 26 O.S. 25	0.20 0.18	2.70 2.08
10	E.M.	42	F	Cortisone Hydrocortisone Prednisone	7/17/51–11/ 1/53 11/ 1/53– 4/30/55 5/ 1/55– 9/12/57	75 50 20	Wide and open with no exceptions	O.D. 18 O.S. 17.5	0.47 0.46	2.88 2.53
11	A.W.	61	F	Cortisone Hydrocortisone Prednisone	5/ 5/51– 9/12/53 9/13/53– 4/24/55 4/25/55– 9/12/57	50 30 5	Wide and open with no exceptions	O.D. 20 O.S. 19	0.23 0.26	1.73 1.74
12	E.K.	50	F	Hydrocortisone Prednisone	4/29/54– 5/ 6/55 5/ 6/55– 9/12/57	40 15	Wide and open with no exceptions	O.D. 18.5 O.S. 18.5	0.45 0.29	2.75 1.77
13	G.K.	69	M	Epinephrine Prednisone	10/10/57–10/17/57 10/10/57–10/17/57	46 injections 100	Wide and open with no exceptions	O.D. 21 O.S. 22	0.18 0.15	1.49 1.35
							Average	20.35	0.24	1.63

Hg (from 13.5 to 26.5 mm. Hg); the average value of outflow facility was 0.24 cu. mm./min./mm. Hg. (from 0.09 to 0.47 cu. mm./min./mm. Hg; the average rate of aqueous flow was 1.63 cu. mm./min./mm. Hg (from 0.60 to 2.88 cu. mm./min.). This compares well with findings in the normal population.

The present investigative results support the opinion that steroid therapy has no definite influence on intraocular pressure of the primary open-angle glaucoma or normal eyes.[4-6] Furthermore, long steroid therapy apparently does not alter the facility of aqueous outflow or the rate of aqueous formation.

Summary

A group of 13 patients with severe asthma with no history of ocular disease was given ocular examinations which included tonography and gonioscopy while on long-term systemic steroid therapy. It was found that there was no significant tendency to elevation of intraocular pressure or impaired facility of outflow in this group.

I should like to thank Dr. John Irwin for his friendly help and co-operation during this investigation.

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MULTIPLE SCREENING FOR EYE DISEASES*

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The screening of large segments of the population for diseases such as tuberculosis, syphilis, and diabetes mellitus has been done for many years, but the use of several tests at one time to detect several diseases (multiple or multiphasic screening) is a fairly recent development.[1,2] The value of mass screening for glaucoma has been emphasized by Foote and Boyce[3] who summarized the results of glaucoma screening projects in this country.

A well-organized multiple screening program as an adjunct to a periodic health examination has been in effect as part of a group practice prepaid medical plan at this hospital since 1951.[4] During the year 1956, all persons over 45 who participated in this out-patient department program were referred to the eye clinic to receive a battery of eye tests for the early detection of glaucoma and other preventable eye disease.

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Procedure

Prior to being seen by an internist on

a subsequent visit, persons requesting a periodic health examination received a multiphasic screening which included tests for urine albumin and blood hemoglobin, a modified glucose tolerance test, a serologic test for syphilis, a chest X-ray study, and a Lead I electrocardiogram. While waiting for a urine specimen to be collected one hour after ingestion of 100 gm. of glucose, persons over 45 were directed to the eye clinic for the series of eye tests. On some evenings when more than 30 persons in this age group presented themselves no more referrals were made.

Visual acuity with and without correction was taken by a technician using a Snellen chart at 20 feet and a Jaeger card at 14 inches. Peripheral vision was measured by a registered nurse on the Harrington-Flocks multiple pattern apparatus in semidarkness. Finally, the ophthalmologist determined the color vision, macular vision, and intraocular pressure, inquired about any previous eye trouble, and made the referral. Color vision was tested with the standard Ishihara color charts, using figures 12, 5, 74, and 2 consecutively. Macular vision was checked with chart 1 (the standard chart) of the Amsler charts. The patient, as he fixed upon the central white dot, was asked whether any of the white lines within the square were wavy, broken, dimmed, or absent. Tensions were taken after topical anesthesia with two drops of Ophthaine (Squibb) with the patient supine on an examining table. A Schiøtz tonometer with a 7.5 gm. weight was used.

All tonometry was done by one physician (M. H.) with the same tonometer. Approximately 20 persons per session were screened in the eye clinic four evenings a week from 6:00 to 9:00 P.M. The same trained personnel conducted their assigned tests throughout the course of the study. The work of the first two weeks was considered practice, and the data obtained were not used for statistical purposes. Persons referred who did not return for re-examination were contacted by letter, telephone, or telegraph. Of the 433 referred during the year, six did not return for further study.

Criteria for referral for ophthalmologic examination. A patient was considered to have failed the test for visual acuity if vision was 20/40 or less in either eye with or without glasses. If the visual acuity was poor and the patient had forgotten his glasses, a pinhole vision was used. Since most of the patients were presbyopic, referral was not made for failure in the near vision test only. On the basis of our personal experience with the multiple pattern field screener, it was arbitrarily decided to refer in the peripheral vision test those who missed two targets in one quadrant, one in each corresponding bitemporal quadrant, one in each homonymous quadrant, or a total of three in both eyes. All with defective macular vision and all with defective color vision other than red-green color-blindness were referred. A tension of 30 mm. Hg was used as the screening level for glaucoma.

Data

The data obtained were recorded in a special form (fig. 1) from which IBM cards were prepared. Diagnoses were coded on the basis of site and type of affection using the Standard Classification of Causes of Blindness.[5] The diagnosis of glaucoma was made by repeated tonometry, visual field tests, gonioscopy, provocative tests, and funduscopy.

The data of the first six months of the study are reported herein. During the second six months the ophthalmologist was withdrawn from the research, and tonometry and color vision testing were discontinued. For this reason it is felt that the results of the second six months are not comparable to those of the first six months and accordingly only brief mention will be made of them.

During the first six months of 1956, the eye screening examination was given to 2,027 persons. Of these, 275, or 13.6 per-

MULTIPHASIC EYE CHECK

FILL IN BELOW ONLY IF NOT STAMPED AT RIGHT

Date

Name

Medical Record Number

Date of Birth (Mo., Yr.) | Sex | Coverage and Group

VISUAL ACUITY (Snellen Chart)
Without glasses: R.E. _____ L.E. _____
With glasses: R.E. _____ L.E. _____
1 ☐ 20/30 or better, each eye
2 ☐ 20/40 or less in either eye

NEAR VISION
R.E. _____ L.E. _____
1 ☐ J-6 or better, each eye
2 ☐ Fails J-6 in either eye

COLOR VISION (Ishihara Chart)
1 ☐ Normal
2 ☐ Red-green
3 ☐ Other

MACULAR VISION (Amsler Chart)
1 ☐ Normal
2 ☐ Deficient

PERIPHERAL VISION (Multiple Pattern Field Screener)
1 ☐ Normal
2 ☐ Deficient
3 ☐ Unsatisfactory

TONOMETRY (Schiotz Tonometer)
Reading: R.E. _____ L.E. _____
1 ☐ Normal in each eye
2 ☐ Elevated in one or both

Any history of eye trouble other than refractive error?
Nature: _____
1 ☐ No
2 ☐ Yes

Is patient now receiving eye treatment?
If yes, for what condition? _____
Name of Physician _____
Address _____
1 ☐ No
2 ☐ Optometrist
3 ☐ Other _____
9 ☐ Not determined

Remarks:
1 ☐ None
2 ☐ Potential disease
3 ☐ Other

REFERRED FOR FOLLOW-UP EXAMINATION
1 ☐ No
2 ☐ Yes - staff physician
3 ☐ Yes - non-staff physician

Follow-Up Examination Diagnosis		Previously Diagnosed: No	Previously Diagnosed: Yes	Code
PRIMARY DIAGNOSIS	Right eye			
	Left eye			
SECONDARY DIAGNOSIS	Right eye			
	Left eye			
PRIMARY ETIOLOGICAL FACTOR RESPONSIBLE FOR ABOVE	Right eye			
	Left eye			

Fig. 1 (Levatin, et al.). Form used in recording data.

cent, were referred to the eye clinic for further examination. Nearly half of this group were found to have conditions other than refractive errors, but about three-fourths of them were previously known. Eighteen persons were found to have glaucoma, and, of these, 15 had not been previously diagnosed. Of the 31 persons with newly discovered conditions, almost half had glaucoma. The glaucoma was all of the chronic wide-angle type (table 1).

Almost half of the newly discovered cases of glaucoma were in the age group 55 to 64, with a rate of 1.0 percent for those screened in that age group. Differences in the age-specific rates shown in Table 2 are not statistically significant, however.

Negroes constituted less than five percent of those screened, but made up 20 percent of the newly discovered cases of glaucoma. The rate of 3.3 percent among Negroes screened is significantly higher than the rate of 0.6 percent among whites. In addition, it was found that the Negroes tested represented a significantly younger population than the whites tested; 55 percent of the Negroes

TABLE 1

SUMMARY OF EYE SCREENING RESULTS BY SEX

Classification	Total*	Males	Females
Number of persons screened	2,027	905	1,059
Number referred	275	153	117
Number with positive diagnosis other than refractive error	*119*	*66*	*52*
Previously known	88	49	38
Newly discovered	31	17	14
Number with diagnosis of glaucoma	*18*	*11*	*7*
Previously known	3	2	1
Newly discovered	15	9	6

* Includes sex not stated.

TABLE 2

NUMBER AND PERCENT OF NEWLY DISCOVERED GLAUCOMA CASES BY AGE GROUP

Age Group (yr.)	Total Screened	Diagnosis of Glaucoma	
		Number	Percent
Total	*2027*	*15*	*0.7*
Under 45	20	—	—
45–49	559	3	0.5
50–54	454	4	0.9
55–59	395	4	1.0
60–64	286	3	1.0
65 and over	210	1	0.5
Age not stated	103	—	—
Median Age	54	55	—

were in the age group 45 to 49, whereas only 28 percent of the whites were in that age group (table 3).

Table 4 summarizes the positive findings of the survey by screening test. If the aim of screening is to refer, insofar as possible, only those people who will be determined on further examination to have definitive diagnoses, tonometry rates high as a screening device. Less than one percent of those tested were referred, and 80 percent of those referred were found to have previously undetected glaucoma. The second most effective device was the macular vision test, which referred four percent, and found a little more than 20 percent with newly discovered conditions. The peripheral vision test referred 4.4 percent. Of these, 34 per-

TABLE 3

NUMBER OF PERSONS TESTED, REFERRED, AND DIAGNOSED WITH GLAUCOMA, BY RACE

Race	Total Screened	Referred	With Glaucoma		Newly Discovered Glaucoma as a Percent of Total Persons Screened
			New	Previously Known	
Total	*2,027*	*275*	*15*	*3*	*0.7*
White	1,870	249	12	1	0.6
Negro	90	17	3	2	3.3
Other	11	1	—	—	—
Not stated	56	8	—	—	—

TABLE 4

SUMMARY OF RESULTS OF VISION SCREENING BY TEST

	Visual Acuity	Color Vision	Peripheral Vision	Near Vision	Macular Vision	Tonometry
Total tested	*2,006*	*2,025*	*2,000*	*2,022*	*2,021*	*1,935*
Referred for follow-up, positive test	162	11[a]	87	100[b]	79	15
No follow-up	2	0	2	2	1	0
No ocular pathology	0	0	49	5	19	2
Diagnosis of refractive error only	74	0	7	26	7	0
With positive findings other than refractive error	86	11	29	67	52	13
Newly discovered condition	15	1	8	10	17	12

[a] Other than red-green color blindness.
[b] Does not include persons who failed only the test for near vision.

cent had a positive finding other than refractive error and less than 10 percent had previously unknown disease. The color vision test referred 0.5 percent. While all those referred for other than red-green color-blindness had a positive finding other than refractive error, each of the conditions was found by one or more of the other tests used and only one of the referrals had a newly discovered disease.

Table 5 deals with the total diagnoses made as a result of referral from the screening examination. This table differs from preceding ones in that secondary diagnoses are included, and shows the relative effectiveness of the various screening tests in detecting specific conditions.

Twelve of the 15 newly discovered cases of glaucoma were positive on the tonometer test. Of the remaining three, two had tensions of 27, and one of 29.5 on the screening test. All of the newly discovered central retinopathies were revealed by the test of macular vision. The test of peripheral vision picked up only four of the 15 cases of glaucoma, and was less effective than any of the other tests, except color vision, in screening the 36 newly diagnosed conditions.

Table 6 shows the result of each screening test for all of the cases of glaucoma found in the survey. The tonometer test detected 10 cases of glaucoma which were not disclosed by any other test. Of the remaining five, two failed tonometry in addition to the other tests, one failed all of the other tests, one failed only the peripheral vision test, and one failed only the macular vision test. The peripheral vision test was positive in four of the 15 cases of newly discovered glaucoma but three of these were detected by two or more of the other screening tests. The multiple pattern test was positive in all cases of glaucoma found by perimetry to have an appreciable field loss.

During the second six months of the survey, 1,881 persons were screened, and 158 were referred for follow-up. Of the latter, 83 were found to have diagnoses other than refractive errors, and in only seven cases were these conditions previously unknown. A case of early retinal detachment was detected by the multiple pattern screener but no previously unknown cases of glaucoma were discovered.

Comment

The glaucoma rate in the population screened was slightly less than one percent. The rates for other screened populations, as reported by Brav and Kirber,[6] Wolpaw and Sherman,[7] Zeller and Christensen,[8] Smillie et al.,[9] and Vaughan et al.,[10] were in the range of 0.4 percent to 2.8 percent. Our finding of one percent may be related to the age composition of the persons screened (median age 54 years) and the high screening level used for tonometry (30 mm. Hg).

The multiple pattern field test was more time consuming than tonometry, required an alertness and rapid reaction time that some patients did not possess, and failed to detect most of the early cases of glaucoma. The Amsler chart was a surprisingly efficient device for detecting relative and absolute central scotomas due to disease of the macula or optic nerve. The charting was done in a minute or two and furnished a permanent record. The central retinopathies discovered consisted of degenerative lesions or scars resulting from old chorioretinitis or macular hemorrhages and were not amenable to treatment.

Summary

During a six-month period, as part of a periodic health examination, 2,027 persons over 45 were given a series of tests consisting of visual acuity, near vision, peripheral vision, macular vision, color vision, and tonometry. Among those screened 275 (13.6 percent) were referred for further examination.

1. Of the referrals 119 (43.3 percent) were found to have a positive diagnosis other than refractive error; 31 of these had newly discovered conditions.

2. Eighteen persons (0.9 percent of the

TABLE 5

SUMMARY OF DIAGNOSES RESULTING FROM SCREENING BY TEST FAILED, FIRST SIX MONTHS

Diagnosis	Total*		Visual Acuity		Color Vision		Peripheral Vision		Near Vision		Macular Vision		Tonometry	
	Newly discovered	Previously diagnosed	Newly discovered	Previously diagnosed	Newly discovered	Previously diagnosed	Newly discovered	Previously diagnosed	Newly discovered	Previously diagnosed	Newly discovered	Previously diagnosed	Newly discovered	Previously diagnosed
Glaucoma	15	3	3	2	1	1	4	3	2	1	3	2	12	1
Refractive error	4	76	4	74	—	1	—	8	2	25	1	8		
Microphthalmos	—	1	—	1							—	1		
Enucleation	—	2	—	2					—	2				
Esotropia	—	5	—	5			—	1	—	5	—	2		
Exotropia	—	8	—	8			—	2	—	8	—	3		
Hyperopia	—	1	—	1					—	1				
Keratitis	—	2	—	2					—	2	—	1		
Keratoconus	—	1	—	—										
Corneal scar	—	11	—	10					—	7	—	2		
Cataract	5	19	3	19	—	1	2	4	1	13	3	3		
Aphakia	—	2	—	1					—	1				
Central retinopathy	10	15	5	12	—	3	1	4	5	9	10	12		
Retinitis (diabetic)	—	1	—	1	—	1	—	1	—	1	—	1		
Chorioretinitis	1	8	1	6	—	2	1	4	1	3	1	4	—	1
Detached retina	—	1	—	1	—	1	—	1			—	1		
Retinal hemorrhage	—	3	—	3	—	1			—	2	—	2		
Optic nerve atrophy	—	3	—	1	—	2	—	2	—	2	—	1		
Hemianopsia	1	1	—	—			1	1						
Amblyopia	—	3	—	2					—	2	—	1		
Nystagmus with amblyopia	—	1	—	1					—	1				
Amblyopia secondary to refractive error	—	2	—	2					—	2	—	1		
Total Diagnoses	36	169	16	154	1	13	9	31	11	87	18	45	12	2

* Rows do not add across because individuals failed one or more tests.

TABLE 6

DIAGNOSED CASES OF GLAUCOMA, NEWLY DISCOVERED AND PREVIOUSLY KNOWN, BY RESULTS OF TESTS

Type of Case and Date of Screening	Test					
	Visual Acuity	Color Vision	Peripheral Vision	Near Vision	Macular Vision	Tonometry
Newly discovered 1–4						X
1–17			X			a
1–24			X		X	X
2–21	X					X
3–7						X
3–7						X
3–28						X
4–2						X
4–6						X
4–17						X
4–24						X
4–27						X
5–8	X	X	X	X	X	a
5–14	X		X	X		X
5–15					X	b
Previously known 1–3	X		X			0
3–23			X		X	X
6–24	X	X	X	X	X	0

X = positive test
Blank = negative test
0 = test not given

a = Tension 27 mm. Hg (Schiøtz)
b = Tension 29.5 mm. Hg (Schiøtz)

total screened) were found to have glaucoma, and, of these, 15 had not been previously diagnosed.

3. A rate of 3.3 percent of newly discovered glaucoma cases was found among Negroes, and only 0.6 percent among whites. Furthermore, 55 percent of the Negroes tested were in the 45 to 49 age group, whereas only 28 percent of the whites tested were in that group.

4. (a) As a result of tonometry 0.8 percent of the total tested were referred, and 80 percent of those referred were found to have previously unknown glaucoma. Of the 15 newly discovered cases of glaucoma 10 were not disclosed by any test other than tonometry, and two more failed other tests besides tonometry. (b) The peripheral vision test referred 4.4 percent and found only 9.4 percent of those referred with previously unknown disease. Only four of the 15 new cases of glaucoma were picked up by the multiple pattern field screener. (c) The macular vision test referred 3.9 percent and

found 21.8 percent with newly discovered conditions. All of the 10 newly discovered central retinopathies were revealed by the Amsler chart.

5. During the second six-month period, tonometry and color vision tests were not done. Among the 1,881 persons screened not a single case of previously unknown glaucoma was discovered.

Conclusions and Recommendations

1. When multiple screening for eye diseases was performed as part of a periodic health examination in a group of patients over 45, the only significant disease found from the standpoint of preventable blindness was chronic simple glaucoma.

2. Tonometry was the most effective and efficient screening test for this disease. The multiple pattern field screener was relatively ineffective in detecting early glaucoma, although it was reliable in detecting more advanced glaucoma with definite field loss.

3. The use of the Amsler chart is highly recommended whenever disease of the macula or optic nerve is suspected.

4. The age level for screening Negro populations for glaucoma should be earlier than for whites (perhaps age 30 years) for chronic primary glaucoma apparently occurs in the Negro at a younger age.

Kaiser Foundation Hospital (11).

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Ophthalmic Miniature

Said a man with a new Schepens scope,
"This thing makes me feel like a dope.
I examine the nose,
The hair and the toes.
See the retina? Some day I hope."

Alan Davidson,
New Bern, North Carolina.

A REGIONAL HEMIRETINAL DIFFERENCE IN AMBLYOPIA*

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In strabismus, with and without amblyopia, different types of suppression occur as a binocular anomaly. The involved areas of suppression are the central part of the visual field and the more peripheral area. The first type of suppression, known as the foveal or parafoveal suppression area, may be plotted monocularly, as an absolute scotoma, in amblyopia with or without strabismus. The second type of suppression, known as the regional hemiretinal (peripheral) suppression area,[1] has been known, heretofore, exclusively as a binocular anomaly. The characteristics of the regional hemiretinal suppression differ in esotropia and exotropia.

It is hypothesized that this peripheral suppression might be revealed as a monocular anomaly in amblyopia, where suppression phenomena are generally enhanced. Furthermore, there might be differences in esotropes compared with exotropes.

BACKGROUND

In esotropia, a nasal hemiretinal area is suppressed, while in exotropia a temporal hemiretinal area is suppressed. Numerous investigations[2] have shown that there are differences in the behavior of the nasal and temporal hemiretinas, based upon developmental, anatomic, and physiologic factors.

Phylogenetically, the big step from separate fields of vision to a binocular field of vision was accompanied by a significant change in the course of the optic pathways. The bundle of the optic nerve which formerly crossed completely in the chiasma, so that one retina was represented in one geniculate body, and vice versa, now divided itself, so that now only the fibers from the nasal sides of the retinas crossed to the other side. The fibers of the temporal retinas remained uncrossed to the lateral geniculate body of the same side. Each geniculate body therefore now contains the ganglion cells of the nasal half and a temporal half of each retina, and this separation continues up to the calcarine fissure. These developments have been accompanied by a radical change in the perceptual mechanisms, because with overlapping of the visual fields, the sensations from both eyes had to be melted together into a binocular perception. This development involves phenomena such as retinal correspondence, horopter differences, and so forth. Most of these ideas are elucidated well by Walls.[3]

The temporal retinas, corresponding to the nasal overlapping visual fields, are less fixed in their manifested functions compared with the phylogenetically older temporal, partly monocular, fields. The embryologic development of the temporal retinas also lags behind the nasal retinas.

Some of the anatomic and physiologic differences between the two hemiretinas occur in the structural distribution of rods and cones (Osterberg[4]), such as the greater frequency of retinal cystic degeneration in the temporal retina, the high incidence of retinal holes and detachments which occur in the upper temporal retinas, photopic visual light thresholds (Sloan), and color sensations.

Another monocular nasal-temporal difference has been noted by Broendstrup.[2] This concerns the poor "nasal light projection" occurring in patients who had a diffusion of

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the optical imagery occurring in one eye for various reasons.

Binocular impressions seem to be molded by those received from the nasal hemiretina, according to Remky,[5] while those received from the temporal hemiretina tend to fade. The temporal retina appears to be inferior to the nasal hemiretina in its binocular functions.

The binocular suppression phenomena occurring in esotropes have been found to differ from those occurring in exotropes. In esotropia the nasal hemiretina is involved in suppression. Its pathogenesis is a rather gradual centrifugal expansion of a central (foveal) suppression into a hemiretinal regional suppression, which may be accompanied by certain abnormalities such as fixation disparity of significant amounts, abnormal retinal correspondence, and eccentric fixation, all of which point to a kind of adaptability of the nasal retina to a new situation, not necessarily as applicable to the temporal retina.

In exotropia the temporal hemiretina is involved in suppression. Its pathogenesis differs from that found in esotropia. All exotropes have developed, in part, a normal binocular fusion status during infancy. The exotropia usually manifests itself at a later age when suppression phenomena have a more "all or none" character. This is well illustrated in patients with intermittent exotropia in whom there is no sensory anomaly when the eyes are straight and fusing, but when one eye diverges the suppression in the involved area is relatively complete.

The purpose of this paper is to show that the binocular peripheral suppression found in strabismus may manifest itself as a monocular anomaly in certain instances. This would be the counterpart to the known monocular foveal scotoma found in amblyopia. All suppression phenomena are enhanced in amblyopia. The binocular regional suppression is deepest in exotropia. It might be expected, therefore, that patients with exotropia and amblyopia would best show such a monocular phenomenon.

Fig. 1 (Nawratzki and Jampolsky). The adapted Harrington-Flocks screener with rheostat.

The usual clinical tests such as peripheral visual fields, as well as peripheral dark adaptation curves, appear to show no differences in behavior of the amblyopic eye compared with a normal eye.

For this investigation another test has been designed to show that there exists a difference of sensation in the nasal and temporal hemiretinas in the amblyopic eye compared with a normal eye. According to Bender,[6] the most sensitive test for visual field examination is the simultaneous stimulation of two points, one in each hemiretina. He used this method of presenting two targets simultaneously for the early detection of visual field defects. Cibis used simultaneous presentation of equal color targets and observed a differential "fading" in different quadrants of the retina.

Harrington and Flocks[7] have constructed a "multiple-pattern field screener" utilizing Bender's principle. This instrument was adapted for our tests (fig. 1).

Method

Equipment

The patient views the screen at the usual distance with one eye occluded. The screen is a white shield upon which the target pattern has been painted with a fluorescent sulfide ink. The screen is

Fig. 2 (Nawratzki and Jampolsky). One of the three test screens, showing the fixation point and the two fluorescent light points, one in the nasal and the other in the temporal field, each at an equal distance from the midline.

illuminated with ultraviolet radiation from a black light tube covered by a tubular Corning #5874, which makes the two fluorescent targets simultaneously visible. A black fixation mark is in the center of the screen.

The screener was adapted so that the ultraviolet light intensity could be controlled by a rheostat (Variac) with a scale from zero to 130, where 110 represents the original (usual) intensity. The screener was used under normal daylight room illumination.

Three different test situations were used. Each test situation presented had two light points, one in the nasal and the other in the temporal field, each at equal distance from the midline. The lower visual fields were selected for the simultaneous comparison of the two light points because preliminary observations seemed to indicate greater differences in this locale (fig. 2).

Test 1. The two light points were each at six cm. from midline, and 15 degrees radially from the fixation point.

Test 2. The two light points were similarly placed at 6.5 cm. from midline, and 10 degrees radially from fixation.

Test 3. The two light points were at a distance of 11 cm. from midline, and 20 degrees radially from fixation. The targets were illuminated by the usual ultraviolet bulb with the intensity increased gradually from zero setting of the rheostat to that setting where the targets were first seen.

Selection of Patients

Nineteen amblyopic patients between the ages of 15 and 50 years (without other ocular disease) were selected for this study.

A patient was considered to have amblyopia when the best correctable vision in one eye was not equal to the 20/20 correctable vision in the fellow eye. Corrected visual acuity in the amblyopic eye varied from 20/40 to finger counting at four feet. All patients had adequate central fixation in the amblyopic eye. Patients who had eccentric fixation, or whose amblyopia precluded adequate fixation, were not included.

The patients were divided into three groups: (1) amblyopes without deviation (seven patients); (2) amblyopes with esotropia (six patients); (3) amblyopes with exotropia (six patients). Comparisons were made with a control group of 11 normal patients who had 20/20 correctable vision in each eye.

Procedure

Each of the three test situations was presented in the described manner. The patient reported the first appearance of a light, and whether at that point one or two lights were seen. If only one light was seen initially, the patient indicated whether it was in the nasal or temporal field. This was termed the "*appearance point.*" The intensity of the light source was then increased until both nasal and temporal field targets were seen. The patient remained steadily fixating, and, without altering the illumination, he then reported when one or both targets disappeared, which was termed the "*fading point.*"

Each of the three test situations was determined three times for each eye. Scatter plots were constructed for the "fading" phenomenon in such a way as to show any difference between the nasal-temporal retinas in the same eye, and to show a difference between the two eyes.

The answers were recorded as *N* (nasal target fades first), *T* (temporal fades first), *O* (both fade simultaneously). In the calculation, *N* was counted as 1, *T* as zero, *O* as 0.5. The sum of all answers was divided by the number of experiments, and this represents the percentage of the "nasal fading first."

The bad eye (amblyopic) was then plotted against the good eye, the ordinate representing the good eye, and the abscissa representing the bad eye. The zero point of the graph indicates that all temporal field targets invariably faded first; 100 on the graph indicates that all nasal field targets invariably faded first; 50 on the graph indicates that there was no preference for nasal or temporal field target fading first. The quadrants of the scatter plot may be divided into A, B, C, and D (fig. 3).

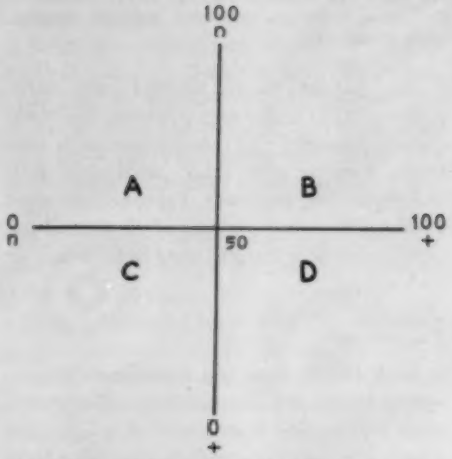

Fig. 3 (Nawratzki and Jampolsky).

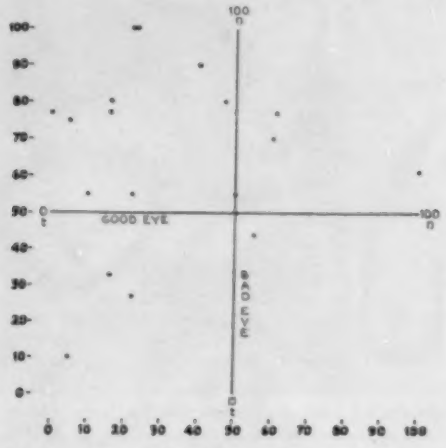

Fig. 4 (Nawratzki and Jampolsky). All amblyopes without glasses.

Points plotted in quadrant (A) indicate that there was a nasal-temporal difference (nasal faded first) and that there was a difference between the two eyes.

Points plotted in quadrant (B) indicate that there was a nasal-temporal difference (nasal faded first) but there was no difference between the two eyes.

Points plotted in quadrant (C) indicate that there was a nasal-temporal difference (temporal faded first) but that there was no difference between the two eyes.

Points plotted in quadrant (D) indicate that there was a nasal-temporal difference (temporal faded first) and that there was a difference between the two eyes.

Therefore, points plotted in quadrants (A) and (D) indicate that there was a nasal-temporal difference and that there was also a difference between the two eyes.

The normal patient group was plotted right eye versus left eye. (Plotting the left eye versus the right eye made no significant difference.)

Patients were examined with and without glasses, and no significant difference was found. Therefore, only data without optical correction have been plotted in the graphs (figs. 4, 5, 6, 7, and 8).

Interpretation

It appears that in this limited series, all patients with amblyopia and exotropia revealed that the targets in the nasal field of the amblyopic eye consistently faded before the targets in the temporal field. This invariable finding was not found in the fellow good eye.

No significant pattern in the nasal or temporal fading in either eye was noted in patients with amblyopia and esotropia, or in patients with amblyopia without deviation, or in normal control group.

Relative to the "appearance point" (not plotted) it is worthwhile mentioning that there was no specific difference found between the two eyes in any group (including normal controls). There was an over-all

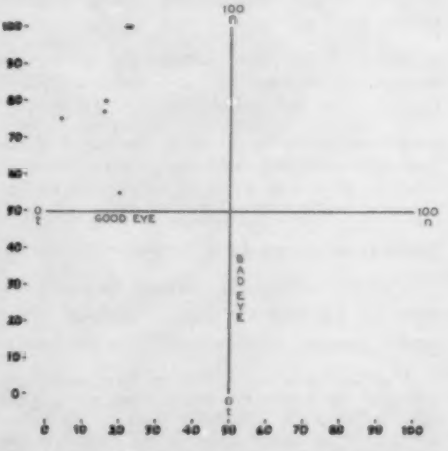

Fig. 5 (Nawratzki and Jampolsky). Exotropic amblyopes without glasses.

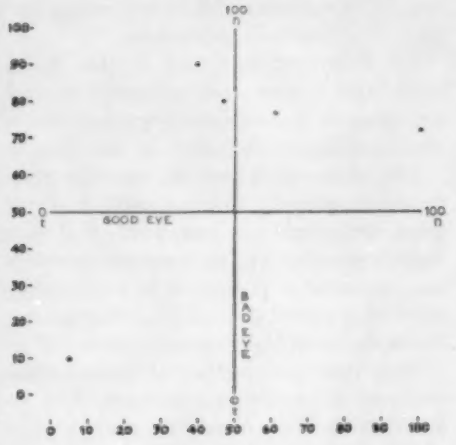

Fig. 6 (Nawratzki and Jampolsky). Esotropic amblyopes without glasses.

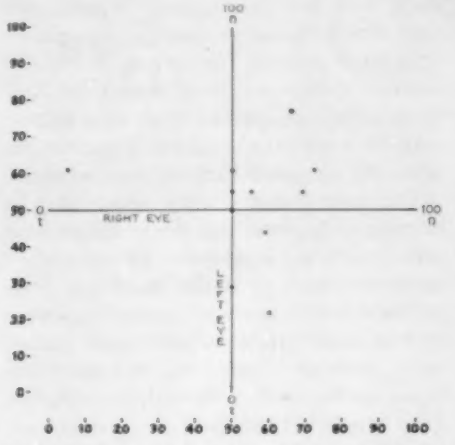

Fig. 8 (Nawratzki and Jampolsky). Normal patients without glasses: Right eye versus left eye.

trend for the temporal field "appearance" before the nasal "appearance."

In exotropic amblyopes there was always a difference in the appearance point between the good and bad eye. In the bad eye, the temporal field target always appeared before the nasal field target. There was very often a remarkable difference in light intensity between the first appearance of the temporal, and the later appearance of the nasal field target. In some instances, the nasal target could not be seen at all, even with the highest intensities available. In these instances, the good eye showed no difference from normal.

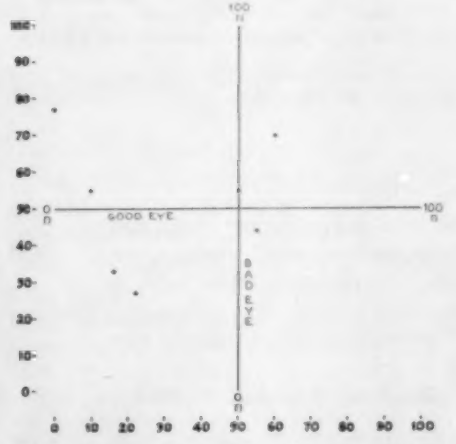

Fig. 7 (Nawratzki and Jampolsky). Amblyopes without deviation, without glasses.

Discussion

The results of this study appear to indicate that the amblyopic eye in exotropia reveals a difference in the nasal and temporal hemiretinas by the tests described. In these patients, the temporal retina (nasal field) appears to be relatively more suppressed than the nasal retina (temporal field).

We wish to call attention to this marked nasal-temporal hemiretinal difference as a monocular anomaly, and secondly, to the marked difference between the two eyes of a pair when this nasal-temporal monocular anomaly is found. This substantiates the working hypothesis that in amblyopia there should be some monocular manifestations of what is ordinarily a binocular peripheral regional suppression in strabismus.

There also appeared to be some general trend for the same type of nasal-temporal difference in all groups (including the normal control group). It is quite possible that the specific technique of the test used in this

study was not adequate to reveal more marked differences in esotropic amblyopes, or in other patients. The very marked nasal-temporal differences found in exotropic amblyopes may be considered as an enhancement of a normal physiologic difference between the nasal and temporal hemiretinas.

The results of this study should not be interpreted to mean that the nasal-temporal difference found is related exclusively either to the exotropia or to the amblyopia. It is probable that it is related to the amblyopia in association with the exotropia. A patient with constant alternating exotropia and equal normal vision showed no variation from normal in either eye.

Summary

The Harrington-Flocks screener was specially adapted to investigate possible nasal-temporal hemiretinal differences in amblyopia with and without strabismus.

All amblyopic exotropes in this series were found to show a marked nasal-temporal difference in the amblyopic eye, and also a marked difference between the two eyes.

This phenomenon was also noted in other amblyopic patients, and in a normal control group, but much less consistently and to a much lesser degree. The nasal-temporal difference found is thought to be an enhancement of a normal physiologic difference between the nasal and temporal retinas.

The hemiretinal regional difference demonstrated in amblyopic exotropes may be considered to be a monocular manifestation of what is ordinarily considered to be a binocular sensory anomaly.

Clay and Webster Streets (15).

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Ophthalmic Miniature

P.S. The Medical Officers would be much obliged to any of the Profession who, in aid of the museum of the Hospital, would send eyes they may hereafter remove, addressed to the Curator, who will be happy to return a report of their minute examination.

N.B.—The eyeball should be put in pure glycerine, immediately after removal.

Ophth. Hosp. Reports, **1**:49, 1857.

THE USE OF PERPHENAZINE* IN OCULAR SURGERY

A PRELIMINARY REPORT

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North Hollywood, California

The success of ocular surgery frequently depends on the effectiveness of the anesthesia and the prevention of vomiting during the operative or postoperative period. For many years there has been a search for the ideal combination of anesthetic agents, and for a drug which will consistently prevent nausea and vomiting. With the advent of the tranquilizing drugs it was immediately recognized that these agents might be of great value in eye surgery because of their ability to relieve anxiety and reduce the incidence of nausea and vomiting.

The most widely used tranquilizer in eye surgery to date has been chlorpromazine. This drug has been used widely in all types of surgery,[1] including eye surgery,[2-6] and has been used in both normal and psychotic patients.[7] The beneficial action of this drug in eye surgery is threefold. First, it relieves anxiety and tension resulting in a more cooperative patient. Second, it reduces the incidence of nausea and vomiting and thus decreases the chance of operative and postoperative complications. Third, it has been well demonstrated that chlorpromazine decreases the intraocular pressure[8-11] and thus provides a softer eye on which the surgeon may operate. This action may help to reduce the incidence of vitreous loss during cataract surgery.

While it is well recognized that chlorpromazine is a valuable drug in ocular surgery, it also has many disadvantages. It potentiates the action of barbiturates, narcotics, and anesthetics so that great care must be taken to make sure that there is not too great a depression of the central nervous system during or following surgery.

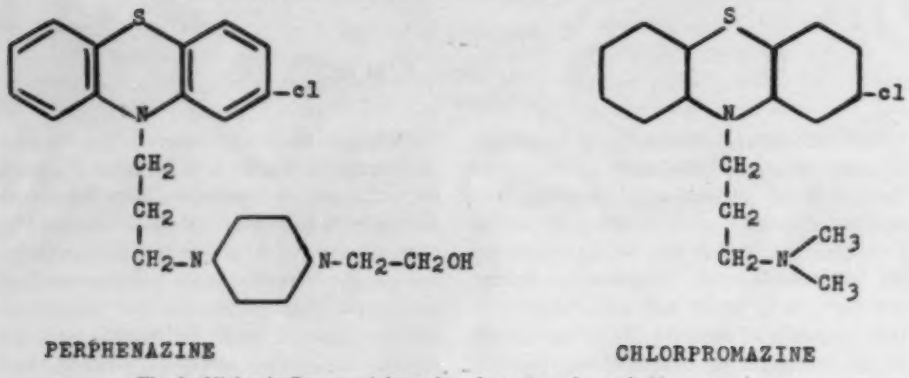
Although their incidence is low, many undesirable side effects have been reported from the use of chlorpromazine. These undesirable side effects include dermatitis, photosensitivity, seizures, jaundice, agranulocytosis, hyperthermia, extrapyramidal symptoms, and effects on the autonomic nervous system such as dryness of the mouth, nasal congestion, constipation, and occasional miosis, or, more rarely, mydriasis. Also chlorpromazine has an undesirable hypotensive effect. Usually this is mild with spontaneous recovery but occasionally this hypotensive effect has been more severe and prolonged, producing a shocklike condition. Since chlorpromazine may reverse the action of epinephrine, the hypotensive state must be treated with nor-epinephrine. In addition, chlorpromazine produces more unwanted drowsiness than any other phenothiazine compound and this may be undesirable in the postoperative period when good patient co-operation may be necessary.

Because of these many disadvantages inherent in chlorpromazine therapy I began using perphenazine (Trilafon) in ocular surgery for its tranquilizing and antiemetic effect. This tranquilizer was first introduced by Schering Corporation for experimental use in 1956. This paper is a preliminary report of my experinece with perphenazine in ocular surgery.

PHARMACOLOGY

Perphenazine, chemically, is 1-(2-hydroxyethyl)-4-(3-[2-chloro-10-phenothiazinyl]-propyl)-piperazine. It is a new amino derivative of chlorphenothiazine. It is obtainable as a white crystalline solid soluble in methanol, ethanol, chloroform, benzene, dimethylformamide, and inorganic acids. It combines with common acids (hydrochloric or maleic) to give crystalline salts which are

* The perphenazine (Trilafon) used in this study was supplied through the courtesy of Harry V. Pifer, Jr., M.D., Division of Clinical Research, Schering Corporation, Bloomfield, New Jersey.



PERPHENAZINE CHLORPROMAZINE

Fig. 1 (Nielsen). Structural formulas of perphenazine and chlorpromazine.

water soluble. Perphenazine has a molecular weight of 403.9 and a melting point between 94-95°C. Upon exposure to light, alcoholic or aqueous solutions of perphenazine become pink. The median lethal dose with perphenazine is much less than that of chlorpromazine. The structural formula of perphenazine is compared with that of chlorpromazine in Figure 1.

Clinical action

Perphenazine is similar to chlorpromazine and may be assumed to act principally on the reticular activating system of the brain stem.[12] The effect of the drug has been assessed on mice, rats, dogs, cats, monkeys, and man. In animals it produces a suppression of locomotor activity and a suppression of avoidance behavior.[13] In man, anxiety, tension, apprehension, agitation, aggression, and fear tend to be alleviated. As a direct result of the broad ataractic action patients become quieter, more relaxed, and less tense, yet generally remain alert throughout the day.

It has also been shown that tolerance to perphenazine develops to a much less degree than to chlorpromazine.[13] Although phenothiazine tranquilizers, such as chlorpromazine, are known to potentiate the action of barbiturates, narcotics, and anesthetics, this action has not been a clinically important factor with perphenazine. Therefore, full doses of barbiturates and narcotics may be given without undue concern about the enhancement of their depressant action. Sedation does not appear to be a problem with perphenazine since patients receiving it in the usual doses are alert and wide awake as contrasted to the drowsiness experienced by patients taking other phenothiazine compounds.[14] Perphenazine has the lowest order of sedation of any known phenothiazine compound. The degree of sedation is: (1) thorazine, (2) pacatal, (3) sparine, (4) compazine, (5) trilafon, beginning with the compound producing the most sedation.

Perphenazine is much more active in its tranquilizing and anti-emetic effects than any of the other phenothiazine compounds. Using chlorpromazine as a standard for comparison, clinical experience indicates that perphenazine is five to 10 times as potent as chlorpromazine. The relative therapeutic potencies of the phenothiazines as a group are shown in Figure 2.

The anti-emetic effect of perphenazine has been reported to be 24 times as active as chlorpromazine,[15] so it is probably the most potent anti-emetic drug available. Nausea and vomiting are thought to be caused by indirect stimulation of the vomiting center via the chemoceptive emetic trigger zone.[16]

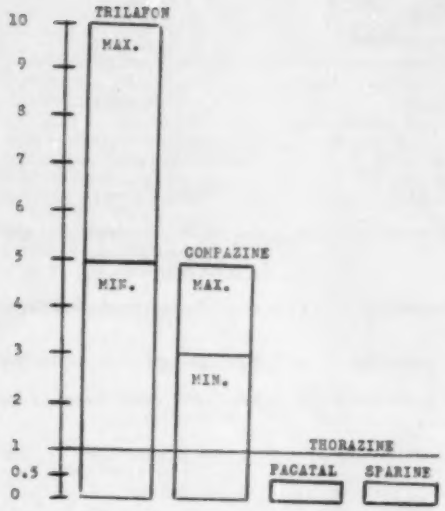


Fig. 2 (Nielsen). The relative potencies of the phenothiazines.

It is believed that the anti-emetic action of perphenazine is accomplished by blocking the action of the emetic agents within the chemoceptive emetic trigger zone and thus preventing activation of the vomiting center.

As stated previously, almost all of the phenothiazine compounds have many undesirable side effects. Perphenazine has been used on many thousands of patients and no serious side effects have been reported to date. It apparently does not cause jaundice, agranulocytosis, photosensitivity, drowsiness, hyperthermia, hypotension, and autonomic symptoms. A few cases of skin rash have been reported. The only side effect of note is the production of extrapyramidal symptoms which is very rare on the usual low dosage regimen. Almost all cases of extrapyramidal effects reported were on high doses and the symptoms were readily controlled by the concomitant administration of anti-Parkinson drugs such as benztropine methanesulfonate. The side effects of the phenothiazine compounds are summarized in Table 1.

Present study

The present study consists of 50 operative procedures in which some of the patients were psychotic and others were normal mentally. In all patients perphenazine was used preoperatively and postoperatively. The dosage schedule used was as follows: Each patient received 4.0 mg. perphenazine four times a day for two days before surgery. Seconal, 0.1 gm., was administered the evening before surgery. Approximately one hour before surgery each patient received 10 to 15 mg. perphenazine intramuscularly along with 0.1 gm. of Seconal and 2.0 mg. Levodromoran. In each case an O'Brien and retrobulbar block was used. The Seconal is necessary because perphenazine alleviates anxiety, worry, tension, and produces a calm patient, but it does not produce drowsiness or sleep. On the above regimen patients arrived in surgery calm, relaxed, and drowsy but would immediately respond when their names were called or when they were asked to co-operate in any way. In almost all patients no greater degree of co-operation could have been desired. The response of the patients so prepared is summarized in Table 2.

Postoperatively the average patient was

TABLE 1

Side effects of phenothiazine compounds

Side Effects	Trilafon	Compazine	Thorazine	Sparine	Pacatal
Parkinsonism	Yes	Yes	Yes	Yes	Yes
Dermatitis	No	Yes	Yes	Yes	Yes
Photosensitivity	No	Yes	Yes	Yes	Yes
Seizures	No	Yes	Yes	Yes	Yes
Jaundice	No	No	Yes	Yes	Yes
Agranulocytosis	No	No	Yes	Yes	Yes
Hyperthermia	No	No	Yes	Yes	Yes
Hypotension	No	No	Yes	Yes	No

TABLE 2

RESPONSE OF PATIENTS

Patient's Initials	Age	Diagnosis	Surgery	Nausea or Vomiting	Comments
E.W.A.	17	Congenital cataracts O. U.	Discission O. U.	0	Usual dosage, good cooperation
L.A.D.	74	After cataract O. S.	Discission O. S.	0	Usual dosage, good cooperation
G.M.	74	Mature cataract O. D.	Cataract extraction	0	Psychotic, received 56 mg. daily postoperatively with a good response
E.W.A.	17	Congenital cataracts O. U.	Linear extraction O. D.	0	Usual dosage, good cooperation
E.H.	67	Immature cataract O. S.	Cataract extraction	0	Usual dosage, good cooperation
P.C.	77	Immature cataract O. S.	Cataract extraction	0	Psychotic, usual dosage, good cooperation
L.J.	82	Immature cataract O. D.	Cataract extraction	0	Psychotic, required 32 mg. daily 5 days postoperatively, good response
L.A.D.	74	Immature cataract O. D.	Cataract extraction	0	Usual dosage, good cooperation
P.M.W.	77	Immature cataract O. D.	Cataract extraction	0	Usual dosage, good cooperation
J.W.E.	70	Immature cataract O. D.	Cataract extraction	0	Psychotic, required small amount of pentothal, good postoperative cooperation
K.S.	70	Immature cataract O. D.	Cataract extraction	0	Psychotic, usual dosage, good cooperation
M.L.	77	Immature cataract O. D.	Cataract extraction	0	Usual dosage, good cooperation
R.S.	66	Lid tumor O. D.	Excision & repair	0	Usual dosage, good cooperation
M.T.	77	Chronic simple glaucoma O. S.	Iridencleisis	0	Usual dosage, good cooperation
D.H.	69	Immature cataract O. D.	Cataract extraction	0	Usual dosage, good cooperation
H.M.R.	74	Apakic glaucoma O. D.	Cyclodialysis	0	Usual dosage, good cooperation
J.R.	67	Immature cataract O.D.	Cataract extraction	0	Usual dosage, good cooperation
A.L.	70	Chronic simple glaucoma O. S.	Corneoscleral trephine	0	Usual dosage, good cooperation
M.L.F.	73	Immature cataract O. S.	Cataract extraction	0	Usual dosage, good cooperation
W.W.S.	28	Congenital cataract O. D.	Discission	0	Usual dosage, good cooperation
K.C.S.	70	Immature cataract O. S.	Cataract extraction	0	Usual dosage, good cooperation
I.D.	70	Immature cataract O. D.	Cataract extraction	0	Usual dosage, good cooperation
I.E.F.	69	After cataract O. S.	Discission	0	Usual dosage, good cooperation
J.A.M.	81	Immature cataract O. D.	Cataract extraction	0	Usual dosage, good cooperation
E.A.	17	Congenital cataract O. S.	Linear extraction	0	Usual dosage, good cooperation
C.F.	47	Mature cataract O. D.	Cataract extraction	0	Usual dosage, good cooperation
W.W.S.	28	Congenital cataract O. D.	Linear extraction	0	Usual dosage, good cooperation

TABLE 2 (*Continued*)

Patient's Initials	Age	Diagnosis	Surgery	Nausea or Vomiting	Comments
M.S.	82	Immature cataract O. D.	Cataract extraction	0	Usual dosage, good cooperation
B.L.	29	Lid tumor O. S.	Excision & repair	0	Usual dosage plus local procaine, good cooperation
T.P.	14	Traumatic hyphema O. D.	Paracentesis	0	Usual dosage, good cooperation
H.M.R.	74	Aphakic glaucoma O. S.	Cyclodyalysis	0	Usual dosage, good cooperation
C.H.S.	38	Perforating injury O. D.	Repair of corneal laceration	0	Usual dosage, good cooperation
S.H.	16	Traumatic hyphema O. D.	Paracentesis	0	Usual dosage, good cooperation
J.R.H.	45	Perforating injury O. S.	Repair of corneal laceration	0	Usual dosage, good cooperation
M.G.	87	Immature cataract O. S.	Cataract extraction	0	Usual dosage, good cooperation
C.M.	83	Immature cataract O. S.	Cataract extraction	0	Usual dosage, good cooperation
I.H.	86	Immature cataract O. S.	Cataract extraction	0	Usual dosage, good cooperation
E.H.	85	Absolute glaucoma O. S.	Enucleation	0	Usual dosage, good cooperation
W.V.	85	Mature cataract O. S.	Cataract extraction	0	Usual dosage, good cooperation
A.C.	32	Corneoscleral laceration with secondary glaucoma O.D.	Enucleation	0	Usual dosage, good cooperation
W.E.	63	Immature cataract O. D.	Cataract extraction	0	Usual dosage, good cooperation
L.S.	84	Immature cataract O. D.	Cataract extraction	0	Usual dosage, good cooperation
L.U.	61	Carcinoma of conjunctiva O. S.	Excision & repair	0	Usual dosage, good cooperation
E.C.	64	Senile ectropion O. U.	Kuhnt-Szymanowski	0	Usual dosage plus procaine nerve block, good cooperation
I.H.	86	Immature cataract O. D.	Cataract extraction	0	Usual dosage, good cooperation
P.O.	86	Immature cataract O. D.	Cataract extraction	0	Usual dosage, good cooperation
W.E.	63	Immature cataract O. S.	Cataract extraction	0	Usual dosage, good cooperation
C.M.	83	Immature cataract O. D.	Cataract extraction	0	Usual dosage, good cooperation
M.A.	86	Immature cataract O. S.	Cataract extraction	0	Usual dosage, good cooperation
W.V.	85	Immature cataract O. D.	Cataract extraction	0	Usual dosage, good cooperation

continued on 4.0 mg. perphenazine three times daily for three days. In psychotic patients, or those who were unco-operative, the dosage was increased until the desired result was obtained. No side effects were evident in my series of patients. One psychotic patient was on 56 mg. a day for one week with no extrapyramidal symptoms or other side effects. If the patient tended to be uncooperative, the perphenazine was continued as long as necessary.

Conclusions

Admittedly, 50 surgical cases is not a large series, but this paper is submitted as a preliminary report, and certain conclusions can

be drawn on the basis of this limited experience.

Scheie has expressed the opinion that the introduction of tranquilizing drugs constitutes one of the most important recent advances in anesthesia for ocular surgery. The tranquilizing, calming, and anti-emetic effect of such drugs has an impressive therapeutic effect on apprehensive, tense, and anxious patients. The unique qualities of the drugs are of tremendous value for the surgeon and patient alike in the operating room and during the immediate postoperative period. It is generally agreed that the use of these drugs results in a significant decrease in the incidence of nausea and vomiting following ocular surgery.

To date the most widely used tranquilizer in ocular surgery has been chlorpromazine. However, it has many disadvantages as compared to the newer tranquilizer, perphenazine. Perphenazine is a much more potent drug, and, at the same time, has comparatively fewer side effects than chlorpromazine or any of the other phenothiazine derivatives. Since perphenazine does not significantly potentiate central nervous system depressants, it is much easier to control. Also the patient remains free from the danger of a hypotensive reaction due to the drug. When used postoperatively to prevent nausea and vomiting, perphenazine does not produce drowsiness but leaves the patient alert and co-operative.

In my series of 50 surgical cases, no side effects were encountered and no patient experienced nausea or vomiting either operatively or postoperatively. Only one patient required pentothal due to lack of co-operation, and this patient was psychotic. I could not have asked any better patient co-operation during surgery. In all instances the drug seemed to ensure a smoother postoperative course.

Summary

An attempt has been made to make a preliminary evaluation of the usefulness of perphenazine (Trilafon) in ocular surgery. This drug was chosen for evaluation because of its many advantages as compared to chlorpromazine (Thorazine). It has five to 10 times the therapeutic potency, has fewer side effects, does not potentiate central nervous system depressants, and does not cause the drowsiness experienced with chlorpromazine.

Perphenazine was used in 50 surgical cases and there was excellent patient cooperation, no side effects, and none of the patients experienced nausea or vomiting while on the drug.

Further investigation may show an improved dosage technique which will make the drug even more valuable. A new series is in progress using the drug in all types of ocular surgery.

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PROPHYLACTIC USE OF NEOSPORIN† FOR DONOR EYES*

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Standard procedures for processing donor eyes at the Eye-Bank Laboratory are as follows:[1]

1. The eye is taken from the bottle in which it arrived and placed in an antibacterial bath.

2. After 10 minutes in the bath, the eye is placed in a fresh sterile bottle containing a cotton dental roll saturated with sterile normal saline to maintain a moist atmosphere. In this bottle the eye is examined to see if it is suitable for corneal graft and, if so, is sent to the surgeon who will use the eye.

3. A culture is taken from the gauze or moisture in the bottom of the original bottle in which the eye arrived, plated on blood agar, and incubated at 37.5°C.

The result of this culture is almost never complete before the eye is used, but the procedure is considered valid, since, if the culture is positive, the organism is identified, sensitivity tests are done and the surgeon is notified immediately. He is also told that the culture was taken before the eye was bathed, and it is our experience that in most cases the contaminating organism is destroyed in the bath.

Until recently the solution in which the eyes were bathed was stainless aqueous merthiolate in 1:5,000 solution with normal saline.

The efficacy of this solution was questioned when we had a case in which a recipient eye became infected after corneal grafting. The culture of the donor eye showed that the eye had been contaminated with Pseudomonas aeruginosa, and the same organism was cultured from the recipient eye.

Although such cases are quite rare, we decided to try a bath of antibiotic solution in place of the merthiolate.

A combination of penicillin and streptomycin in liquid paraffin is routinely used for donor eye storage at the East Grinsted Eye-Bank in England,[2] but, because of our experience, we wanted an antibiotic which was effective against Pseudomonas. Hallett, Wolkowicz, and Leopold,[3] in 1956, found that Neosporin was effective against a wide range of bacteria, including Ps. aeruginosa, and Thygeson[4] of San Francisco was using

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* From the laboratory of The Eye-Bank for Sight Restoration, Inc., Manhattan Eye, Ear, and Throat Hospital. Aided by a grant from the Lillia Babbit Hyde Foundation.

† Neosporin® Ophthalmic Solution. We are indebted to Dr. E. N. Whitman of Buroughs-Wellcome, Tuckahoe, New York, for the generous supply of Neosporin used in these experiments.

it for preoperative washing of donor eyes. We therefore decided to compare Neosporin with merthiolate as a bath for donor eyes.

Methods and Materials

Staphylococcus aureus is the organism which was most frequently cultured from donor eyes; Ps. aeruginosa is among the most virulent and difficult to control, so these two organisms were chosen for these experiments.

The staphylococcus cultures used were obtained from the Pathology Laboratory of Manhattan Eye, Ear, and Throat Hospital. All were hemolytic and all were cultured from eyes.

The strain of Ps. aeruginosa was P. 28. Efforts to test other strains failed because, although several strains were sent to us from other laboratories, all but P. 28 died in transit.

Pure cultures of the above organisms were diluted in strengths from 1/10 to 1/1,000,000. Enucleated cats' eyes were immersed in the bacterial baths for one minute and each was then transferred to a bath of merthiolate (1:5,000), Neosporin, or normal saline.

The sequence was as follows:

1. One minute in the bacterial solution.
2. Immersion in antibacterial bath for 10 minutes.
3. Saline wash for two minutes.
4. Eye discarded.
5. Culture taken from the saline wash and plated on blood agar.

After 48 hours' incubation the colonies were counted.

A total of 70 eyes were processed in this way, testing the antibacterial agents against each of the two organisms five times.

The results were quite uniform for all of the runs, and the average of colonies counted were as in Table 1.

TABLE 1

Dilutions	Neosporin	Merthiolate	Saline
Staphylococcus aureus			
1/10	Overgrowth	Overgrowth	Overgrowth
1/100	12	Overgrowth	Overgrowth
1/1,000	3	15	Overgrowth
1/10,000	1	8	14
1/100,000	0.4	3	9
1/1,000,000	0	0.7	3
Pseudomonas aeruginosa			
1/10	Overgrowth	Overgrowth	Overgrowth
1/100	Overgrowth	Overgrowth	Overgrowth
1/1,000	10	12	Overgrowth
1/10,000	4	8	15
1/100,000	0	4	12
1/1,000,000	0	2	4

Comment

Human donor eyes are ordinarily enucleated under sterile conditions, irrigated copiously with sterile normal saline or bathed in antibiotic solution and then placed in sterile bottles. Bearing these precautions in mind, it is impossible that a human donor eye would be grossly contaminated.

The high degree of efficacy of Neosporin in the weaker dilutions of bacteria emphasizes the importance of immediate irrigation of the enucleated eye with sterile normal saline, since the mechanical effect of washing the eye is very valuable.

The results of these tests indicate that Neosporin is an effective antibacterial agent against the two organisms tested and was more efficacious than merthiolate, the previously used solution.

In practice, it must be remembered that the recipient eye is also a possible source of contamination. In the experience of The Eye-Bank, positive cultures of the bottles in which donor eyes arrive are becoming fewer and fewer as hospitals become more familiar with The Eye-Bank and the techniques for procuring suitable donor eyes.

Conclusions

1. Neosporin was tested as an appropriate solution for bathing Eye-Bank eyes. It was found to be highly effective against the weaker dilutions of Staphylococcus aureus and Pseudomonas aeruginosa, the two organisms used in this series of tests. It was found to be superior to merthiolate, the solution previously used.

2. Irrigation of the enucleated eye with sterile normal saline is believed to have an important mechanical effect, washing possible contaminants from the eye.

210 East 64th Street (21).

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ADAPTATION TO GLARE*

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In the large material on glare and visual acuity, the glare effect is usually expressed in terms of properties of the glare source and its location in the visual field. In any physiologic stress situation, however, the time is an important parameter. It was, therefore, desirable to study glare effects on visual acuity with time of exposure as the critical parameter of measurement.

Method

In principle, the method consists in the recognition of fine details (small wires) under continuous glare exposure. Adaptation to glare is expressed as time course of visual acuity.

Twenty wires ranging from 0.7 to 0.0145 mm. were mounted across circular openings of 21-mm. diameter, on two interchangeable discs with 10 openings. Each wire was placed across the opening in a different direction. The visual field presented by the disc openings equalled seven degrees. A 100-watt frosted lamp was fixed centrally on the back side of the box so that the top of the bulb was viewed through the tube and the disc opening. In order to center the openings of the disc correctly for each exposure, the angular positions of the handle corresponding to the centers of the openings were marked on the outside of the box.

For determination of the instantaneous glare effect immediately on exposure the following procedure was used. The eye was exposed to the glare of a 100-watt lamp for the duration of one second. The subject reported whether the wire was visible in its full length, visible only at the ends, or not visible at all. If visible, the subjects were requested to describe the direction of the wire, as a control for the reliability of the measurement. After presentation, a rest interval of 15 minutes was given to recover from the glare effect. Then the measurement was repeated with another wire. Usually, in the first measurement a wire was presented which could be recognized by all subjects in its full length, and in the second measurement a wire was shown which probably could not be recognized. In the subsequent presentations, this range was narrowed down until the thresholds for full recognition and for partial recognition (ends

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of the wire) were established. These thresholds were rechecked in repeat determinations. Frequently, 10 presentations or more were necessary to establish the threshold values, amounting to a total testing time of about four hours. The finest resolving power under glare was expressed in terms of seconds visual angle, for full recognition as well as for partial recognition of the wires.

In most experiments, the level of partial recognition was actually obtained. However, in some experiments, after the last level of full recognition, the next finer wire could not be seen. Obviously, partial recognition was assumed to have occurred at the arithmetic mean between the two levels. In a few experiments, partial recognition was obtained on two consecutive steps of visual acuity. Here, the finest level was taken as criterion. In view of the time consuming procedure of measurement of the instantaneous glare threshold, it was determined in only three subjects.

The course of glare adaptation was determined as time (seconds) for recognition of two wires, corresponding to visual angles of 18 seconds and of 36 seconds, respectively. The time for recognition of the ends of the wire and of the wire in its full length was measured by means of a stop watch. Since both readings are closely related, only the latter is reported. The sequence of presentation was varied at random. Before the actual measurement, a trial experiment was made in order to familiarize the subject with the procedure. The subject rested a few minutes before the actual experiment at a dim surrounding illumination of one foot-candle, which is sufficient for adaptation to this level of brightness. Usually, both eyes were examined, separated by an interval of five to 10 minutes. This interval is safe to exclude interaction, since, according to Mandelbaum,[1] simultaneous exposure of one eye to bright light does not change the light threshold value of the other eye.

Seventy-one experiments with visual acuity level of 36 seconds, and 76 experiments with the acuity level of 18 seconds, were performed in 15 subjects who were familiar with visual testing from previous experiments with dark adaptation.

Results and Discussion

A fine wire, running across an opening, viewed against a bright glare source placed behind that opening, cannot be seen in the first moment of glare exposure. After some time, however, the ends of the wire can be recognized, so that the middle part of the wire appears to be interrupted. Finally, the wire will be recognized in its full length.

The magnitude of the glare adaptation phenomenon can be estimated from comparison of the width of the wires which could be seen immediately after start of the glare exposure and after adaptation.

On the average of four determinations in three observers, the width of the wire seen in its full length on initial glare exposure was 340 sec. and 282 sec. for the ends of the wire. Due to the rapid change of visual acuity under glare and large interindividual differences, the method for measurement of the instantaneous initial glare threshold was not practical for determination of the visual acuity threshold in given time intervals during glare adaptation. Therefore, the finest resolving power under glare adaptation was not actually determined.

All observers were, however, able to see the wire of 18 seconds width in its full length after adaptation, and it is likely that a somewhat thinner wire would have been recognized. The minimum adaptation effect is, therefore, 340:18 corresponding to improvement of visual acuity under continued glare by nearly 20 times. The magnitude of the adaptation phenomenon is large enough to be of practical importance.

The average recognition time of 15 subjects for the wire of 36 sec. width in its full length was 15.2 sec., with a standard

deviation of ±7.46, and for the wire of 18 sec. width it was 32.2 sec., with a standard deviation of ±20.71.

On the average, it takes about twice as long to recognize the wire in its full length than the ends of the wire. The interindividual differences in the recognition time were considerable, as shown in the large standard deviations, but, in general, the glare adaptation is quite rapid, for most subjects being completed within one minute, under the conditions of the experiment. It is likely that brightness of the glare source and properties of the test object will be of importance. The main result, therefore, is the demonstration of the existence of adaptation to glare, which could not be predicted from the continuous drop of light sensitivity under glare.[2] The adaptation effect to glare is not easily compatible with the concept of glare being due to a photochemical retinal process and supports the concept of a neural component.

An interesting phenomenon is the appearance of the ends of the wires before the full length is seen. This was uniformly reported by all subjects, and did not depend on the orientation of the wire. A differential regional functional sensitivity may be involved, perhaps due to the decrease of the number of cones in the visual field of six or seven degrees, as compared to the fovea.

Time course as well as extent of the glare effect are likely to be of practical importance in situations where exposure to glare exceeds 20 seconds. A foveal adaptation effect to glare was found by Schouten and Ornstein,[3] using a revolving disc with two open sectors, one for exposure of the glare source from 0.02 to 0.2 sec., and the other for exposure of the test object for a duration of 0.01 sec. Exposure of glare source and test object was separated by a short interval. The sensitivity was lowest at 0.05 sec. and back to the original level at 0.15 sec. of glare exposure, but the differences were comparatively small. The experimental procedure, time course, extent of adaptation, and size of visual field in Schouten and Ornstein's and in this series are so different that the adaptation phenomenon involved is probably not identical.

Summary

The instantaneous deterioration of visual acuity, determined by means of recognition of fine wires, under glare recovers gradually during the first two minutes of continued glare exposure. This adaptation effect was estimated from the difference of the initial instantaneous visual acuity threshold and an arbitrary final threshold of 18 sec. visual angle which could be recognized by all 15 subjects. It approximated an improvement of 20 times the initial acuity threshold. The adaptation effect could not be predicted from the continuous decline of light sensitivity under glare. There was a large range of individual variability in the speed of glare adaptation.

The Medical School (14).

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THE EFFECT OF IRON COMPOUNDS ON RABBIT VITREOUS*

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In a previous paper on simulated vitreous hemorrhages in rabbits,[1] it was shown that homologous whole blood was capable of liquefying the vitreous, although there was accompanying strand formation and pigment deposition. It seemed important to us to determine the mechanism of this liquefaction, as such a study might provide a simple and noninflammatory method of liquefying vitreous. It was also of interest to investigate why blood does not form a fixed clot in the rabbit's eye while often doing so in the human eye.

To attack these problems we have conducted experiments designed to identify the component in blood which causes this liquefaction and to suggest a possible mechanism for its action.†

MATERIALS

1. GLOBIN

Globin was prepared by the method of Anson and Mirsky.[2] Washed, laked red blood corpusles were cooled to 0°C. and added gradually to a tenfold volume of acetone containing one-percent concentration of HCl (also cooled to 0°C.). This splits the globin from the heme which remains in solution. The mixture is allowed to stand two or three minutes and is filtered, washed with acetone, and dried, keeping the temperature near 0°C. The acid denatured globin is ground in a chilled mortar with water until a maximum amount dissolves. The solution is then carefully treated with 0.2 N NaOH until a slight permanent precipitate is formed. The solution of native globin is then filtered from the denatured protein, still in the cold. One batch of globin was further purified by salting out any denatured protein with 40-percent saturated ammonium sulfate. The native globin was then salted out at 50-percent saturated ammonium sulfate, dissolved, and dialyzed. Usually extraneous precipitates formed during the process and were removed. Final concentration was about 12 mg./ml., as against 135 mg. in the blood in the form of hemoglobin, and attempts to concentrate it resulted only in further denaturation.

2. HEME

Heme was precipitated from the acid-acetone solution (see under "Globin") by adding twice the volume of water.[2] It was then filtered, washed with water, and suspended (with some dissolution) in 1.0 M K_2HPO_4 to approximate the blood concentration of heme in the form of hemoglobin.

3. HEMOGLOBIN PREPARATIONS

A. Fraction II was prepared from fresh oxalated beef blood. The red blood cells were spun down, separated from the white cells, washed three times with 0.9 percent NaCl, and laked in an equal volume of distilled water. After the stroma were separated by centrifugation the solution was brought to 50-percent saturation with a saturated solution of ammonium sulfate. The small precipitate that formed was discarded and the solution was made to 70-percent saturated ammonium sulfate. The precipitate was washed once or twice with 70-percent saturated ammonium sulfate, dissolved, and dialyzed in the cold at least 24 hours against four or five changes of distilled water. The hemoglobin solution was then concentrated to its original volume by dialysis against polyvinyl-pyrrolidone. This solution of

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† A preliminary report was given before the Western Section of the Association for Research in Ophthalmology, November, 1956, San Francisco, California.

Fraction II was made isotonic with NaCl prior to injection.

B. Fraction II was further purified by column chromatography on a cation exchange resin, generally Amberlite IRC-50.[3] XE-64, a finer mesh form of IRC-50, was also used, but the hemoglobin proved to be more difficult to elute from this. Using a citrate buffer of pH 5.81, sodium ion concentration 0.34M, 14 mg. of Fraction II was loaded on an 18 by 180 column, or 140 mg. on the 45 by 230 mm. column. Running the buffer through at a rate of one to two ml./hr. eluted most of the hemoglobin in the fourth and fifth hour on the small column. These fractions were dialyzed against distilled water, concentrated against polyvinylpyrrolidone to their equivalent blood volume, and used for injection. Spectrographic examination* showed this material to be hemoglobin of which 80 percent was in the reduced state.

Methods

An amount of 0.1 ml. of material to be evaluated is injected with a 30-gauge needle into the vitreous of albino rabbits through the pars plana; 0.1 ml. of aqueous humor is withdrawn from the anterior chamber prior to the vitreous injection. The rabbits are then observed for two weeks at which time they are killed and the "liquidity" of the vitreous is estimated. This estimation is based on the amount of fluid which will flow out through a five-mm. scleral window in three seconds. Since the average vitreous weight in the rabbit eye is one gm., the amount liquefied may be considered as a fraction of the whole.

It has been our experience that a slight amount of nonspecific liquefaction is associated with a number of compounds which produce a slight inflammatory response in the vitreous. To rule out these cases we have set the lower limit of significant liquefaction at 0.6.

* Courtesy of Dr. Louis A. Strait, Spectrographic Laboratory, School of Pharmacy, University of California Medical Center, San Francisco.

TABLE 1

Condition of rabbit vitreous 14 days after injection

Fraction Injected (0.1 ml. blood)	Total No. Eyes	Significant Liquefaction (vitreous more than 0.6 liquid)
Plasma	5	0
Rabbit laked red cells	4	3
Beef laked red cells	7	7
Human laked red cells	6	1 (3*)
Fraction II (8 different batches)	29	23
Armour's hemoglobin	7	0 (6*)
Fraction II oxidized (Methemoglobin)	3	3
Fraction II reduced (Hemoglobin)	2	2
Fraction II lyophilized	4	2
Fraction II purified by column chromatography	11	9
Globin (conc. = 0.72 mg./ml.)	10	1
Heme (dilute suspension)	2	0
Cytochrome C	2	0

* Indicates the number of eyes with a massive vitreous exudate.

Results and Discussion

It can be seen from Table 1 that the component which liquefies the vitreous of rabbits is associated with the saturated ammonium sulfate Fraction II of lysed red cells. This fraction is essentially hemoglobin and when it is purified by column chromatography there is much less strand formation and pigment deposit.

Five of the eyes were completely clear or only slightly hazy. The state of oxidation of the iron has little effect. It is apparent that hemoglobin is responsible for the liquefaction of rabbit vitreous.

Since hemoglobin is not ordinarily considered to have lytic properties, it was felt that further investigation into the mechanism of its action was necessary.

When the prosthetic group is separated from the protein the liquefying ability is lost. It should be pointed out that the separated components of hemoglobin, globin,

and heme are much less soluble than the parent compound and the lack of effect is probably due to inability to get enough material into the vitreous. When the heme is degraded further, it was found that the lytic ability resided in the iron molecule. Most of the low molecular weight iron compounds (ferrous and ferric ammonium sulfate, ferric chloride, ferric citrate, ferric versenate, ferric oxalate, and ferrous lactate) would liquefy the vitreous, although better results from the point of view of less pigment deposits and strand formation were obtained with the organic iron compounds rather than with inorganic iron.

From such results it would be plausible to consider that the iron activates a lytic system which liquefies the vitreous. The two-week period for this reaction to occur is in support of such an indirect effect. The iron must be available in sufficient but not too great a quantity for a long enough period of time. Hemoglobin, by its breakdown in the vitreous, fulfills these conditions by gradually releasing iron. Cytochrome C, on the other hand, because it is not metabolized in the vitreous, does not furnish any iron. It diffuses out of the vitreous and leaves it unchanged. Inorganic iron produces considerable strand formation because of its initial presence in high concentration. On a mole for mole basis the injected hemoglobin contains about 0.8 μM of iron which is comparable to the level (0.4 μM) at which the low molecular weight organic iron compounds are effective.

Preliminary experiments seem to show that this mechanism holds only for the rabbit and not for the cat or dog.

Other inorganic and organic compounds were injected into the vitreous. Of the inorganic ions (copper, gold, cobalt, manganese, zinc, aluminum, and ruthenium), only copper produced liquefaction. Copper, either in the ionic form or complexed with glycine, produced a clear liquefaction which was accompanied by almost complete destruction of the retina.

Summary

The component in blood responsible for liquefaction of rabbit vitreous bodies appears to be the iron molecule.

If iron is present in proper concentration for a long enough period of time, liquefaction results. Hemoglobin and certain organic iron compounds fulfill these conditions. The iron hypothetically activates a lytic system which causes the liquefaction of the rabbit vitreous. This is not a general mechanism but seems restricted to rabbit vitreous.

Only copper, of several other ions tested, produces liquefaction, but this is accompanied by retinal damage.

University of California
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OCULAR LEPROSY*

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In nonendemic temperate zones leprosy is rarely seen.[1] Since 1900, only 14 cases have been recorded in The Johns Hopkins Hospital files. In only one of these was an ophthalmic consultation obtained and this was for an unrelated fundus lesion. A review of the clinical records of these patients, however, suggests that leprous ocular involvement was present in several of the other cases. There was certainly a low index of suspicion for leprous eye lesions; moreover, the patient may be first seen with the late manifestations of ocular leprosy. The classical signs of the disease are frequently absent. The examination of the eyes at this stage would not suggest the underlying cause. The following case of hitherto undiagnosed advanced lepromatous leprosy is therefore presented to illustrate some of the features of ocular leprosy and to emphasize the granulomatous ocular changes which are typical in the advanced disease.

F. W., J.H.H. #759656, a 58-year-old white man, native of Texas, complained of blurred vision for two years. For the year preceding admission, he had been treated with local and systemic corticoids for a bilateral granulomatous uveitis.

The family history was noncontributory. The past history disclosed the following features. The patient had daily contact with a group of Chinese laborers during 1917. For 30 years, he had a perforated septum attributed to surgical trauma. For at least 20 years, he suffered generalized paresthesias with anesthetic areas over his extremities. For as long as he could remember he said his eyes had been red.

General physical examination showed a dry and scaly skin with patches of depigmentation, erythema, and necrosis. Elsewhere there were areas of atrophy and scarring. There were several subcutaneous nodules along the lateral aspect of the left upper arm and dorsum of the right fourth finger. A perforated nasal septum and epistaxis associated with a maxillary sinusitis were present. Neurologic examination disclosed a generalized hypesthesia to pain, touch, and temperature. There was thickening of the ulnar nerves associated with atrophy of the intrinsic hand muscles.

Ocular examination showed the right vision reduced to hand movements and the left vision 20/70. The lids, cilia, lacrimal apparatus, and extraocular movements were normal. There was intense hyperemia of the episcleral tissues and bulbar conjunctiva. The corneas of both eyes showed superficial peripheral opacities which resembled an advanced arcus. In the superior quadrants of both corneas, there were superficial nebulas. There was moderate corneal edema. Both anterior chambers were half filled with a greasy purulent exudate. The keratic precipitates were of the mutton-fat variety. Koeppe nodules were numerous and a stromal nodule was noted in the left iris. There were extensive anterior and posterior synechias and secondary lens changes in both eyes. The right fundus could not be seen. Poor visualization of the left fundus disclosed no abnormalities. The tension in the right eye was 72 mm.Hg; in the left eye 55 mm.Hg on the Schiøtz scale. Under Diamox and mydriatics the tension of the left eye fell to normal. The tension of the right eye yielded to paracentesis,

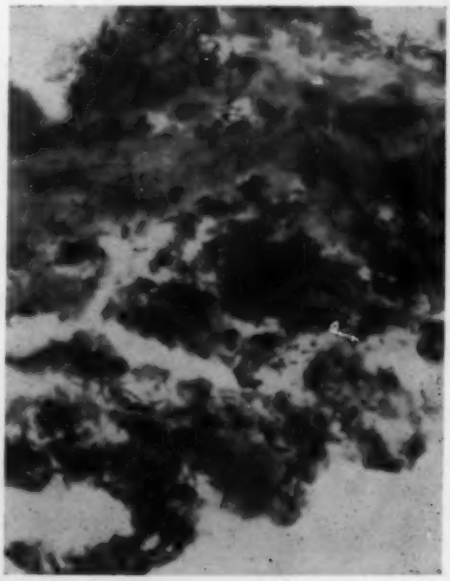

Fig. 1 (Sears). Leprous iritis. Dense bacillary infiltrates and necrosis (Kenyoun stain, ×1,000).

* From the Wilmer Ophthalmological Institute of The Johns Hopkins Hospital. I wish to express my appreciation to Chapman H. Binford, M.D., Chief of the Laboratory of Investigation of Leprosy at the Armed Forces Institute of Pathology, for his consultation.

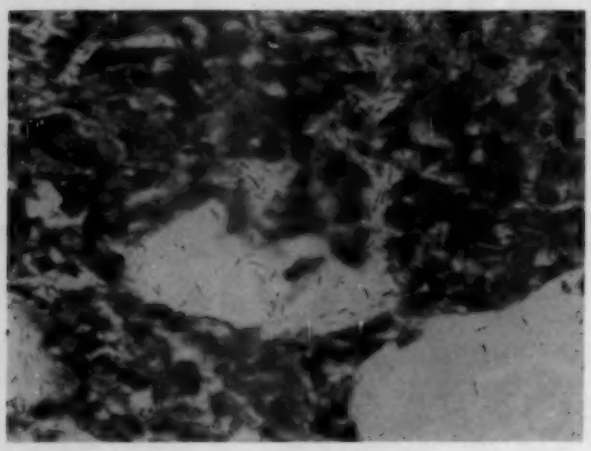

Fig. 2 (Sears). Subcutaneous nodule. Diffuse infiltration of lepra bacilli, intra- and extracellular (Kenyoun stain, ×980).

iridectomy, and irrigation of the anterior chamber.

The usual laboratory and sensitivity studies were unrevealing. Material for biopsy was then obtained from the iris, subcutaneous nodules, and nose. These tissues all showed an infiltrative granulomatous reaction with numerous acid-fast bacilli, characteristically arranged in packet fashion, both intra- and extracellularly (figs. 1, 2, and 3).

One of the most striking features of ocular leprosy is the predilection of the disease process for the anterior ocular segment.[3-4] Only rarely are bacilli or lesions found posterior to the ciliary body. The early clinical manifestations are an episcleritis, secondary conjunctivitis, and superficial keratitis. As the disease progresses, the corneoscleral roll develops from the early limbal lesion which spreads round the cornea, posteriorly into conjunctiva and sclera, and anteriorly into the corneal stroma. Still later the uveal tract is involved. The iritis is often characterized

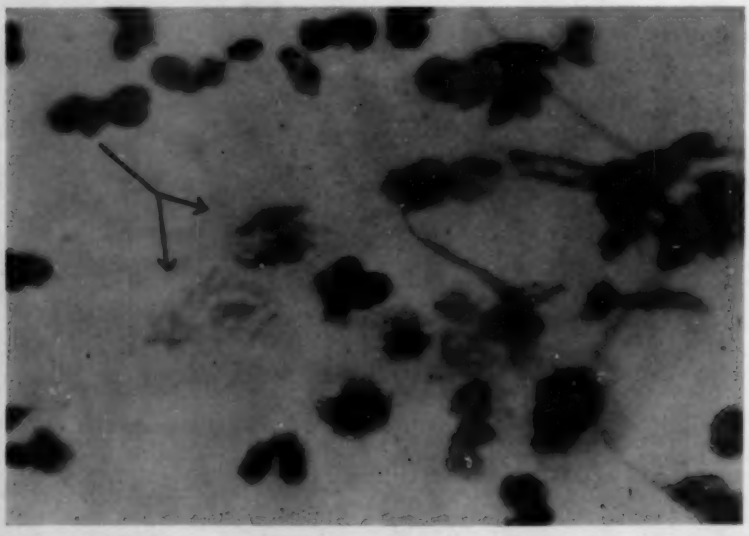

Fig. 3 (Sears). Scrapings from nasal mucosa. A packet of lepra bacilli (Ziehl-Neelson stain, ×980).

by miliary lepromas, small almost pedunculated nodules on the anterior iris surface. DeBarros[5] and Elliot[6] describe a more diffuse type of reaction as well, marked by edema of the iris and gelatinous exudate. Finally the full-blown granulomatous uveitis evolves.

Elliot's analysis[6] of the pathogenesis of leprous eye lesions indicates that the nodular type of uveitis occurs after the leprous patient has harbored the bacillus in the tissues for at least 10 years. The subsequent evolution of the advanced granulomatous uveitis requires still more time. Those who have had experience observing the early development of the disease among infant and adolescent contacts[6] stress microscopic changes in the limbal capillary bed, perineural changes in the corneal nerves,[7] and wispy fibrinous exudates on the iris.

In this particular case of histologically proven leprous uveitis, there was evidence of a previous longstanding superficial keratitis and scleritis. After a prolonged assault, the anterior portion of the uveal tract was affected. Finally the disease took the form of a granulomatous uveitis with Koeppe nodules, edema of the iris, and a marked exudative reaction. Thus, the clinical picture in this late stage masked the characteristic leprous lesions. Although there were elements suggestive of ocular leprosy, not until systemic changes were uncovered, a biopsy obtained, and the bacilli demonstrated was the final diagnosis reached.

The Johns Hopkins Hospital (5)

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INVESTIGATIONS ON THE STRUCTURE OF THE VITREOUS BODY BY ULTRASOUND*

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The vitreous body consist of two parts: the vitreous humor and the insoluble residue. The vitreous humor has been found to contain proteins, albumin, and globulin.[1, 4, 20, 21] The insoluble protein is composed mainly of a collagenlike substance.[15–17, 24] The insoluble protein residue contains carbohydrate, whereas, hexosamine has not been found.[9, 17] In addition to the proteins already mentioned, hyaluronic acid and particularly the interrelation of hyaluronic acid and collagen, are supposed to be significantly important for the gel properties of the vitreous body.[22] It has also been found that the vitreous fibers and one or more polysaccharides associated with them, which contain approximately equal amounts of glucose and galactose, are responsible for the physicochemical properties of the vitreous.[8] Furthermore, a certain amount of a polyuronide which can be separated by incubation from hyaluronidase is associated with the fibers.[8]

In recent years the insoluble protein has been investigated by phase-contrast and electronic microscope and found to contain various fibers,[18, 19] hyaloid membranes, and sheets of fine fibrous material.[2, 6] These fibers have been seen in the vitreous of the cow, the ape, and man. Also when explor-

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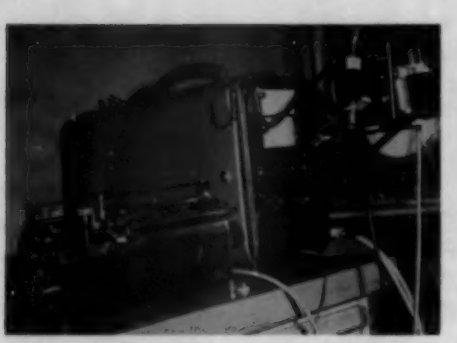

Fig. 1 (Oksala and Lehtinen). The ultrasonic apparatus with the interferometer at the left. To the right the crystal is seen at the end of the cable.

ing the living eye by slitlamp, it is possible to distinguish fine fibers and membranes.[5,8,23] The vitreous body has, therefore, proved to be of a highly differentiated structure, both macroscopically and microscopically, an opinion which was advanced by numerous investigators as early as during the last and at the beginning of the present century.

In a previous paper of ours,[11] in which we studied the velocity of the sound in the vitreous of a bovine eye, we noted that a broken vitreous was not homogeneous acoustically. By means of the ultrasonic equipment, feeble echoes from certain directions in the interior of the vitreous could be registered on the scale of the Brown tube. In the study, we have primarily investigated, by means of an ultrasonic apparatus, the acoustic properties of both unbroken and damaged vitreous bodies, which might permit us to gain some new knowledge about the structure of the vitreous.

Research equipment and material

For our investigations we used the West-German ultrasonic equipment constructed by Dr. J.u.H. Krautkrämer, *Gesellschaft für Elektrophysik*, Köln, which was originally intended for testing of materials in the metal industry (fig. 1). The photograph shows the research equipment on the right with the searching unit at the end of the cable. To the left the interferometer is seen which we have used in our studies on the velocity of sound. The mechanism of action of this equipment has already been described in detail by Mundt and Hughes,[10] as well as by Oksala and Lehtinen.[11]

The subject of our research was the vitreous of the bovine eye. The eyes were examined shortly after slaughtering. It has not been possible to obtain a normal vitreous body of the human eye for this investigation. For several reasons the vitreous of the enucleated eye proved unsuitable, chiefly on account of its extensive pathologic changes. The vitreous of the bovine eye is anatomically similar to that of the human eye,[14] but, owing to its larger size and higher viscosity, it is a better subject for research with the ultrasonic apparatus than the vitreous body of the human eye.

Methods of research

The structure of the vitreous in the bovine eye was investigated by means of ultrasound in two different environments: (1) in the test tube and (2) while the vitreous was contained in the eye, in situ.

For the test-tube studies the vitreous, together with the lens, was carefully detached from the eye without breaking. Thereafter the lens and capsule, still adhering to the vitreous and the pigment of the ciliary body which was detached from its base, were removed from the vitreous by snipping off with scissors. The perfectly transparent vitreous was decanted into a wide test tube and the same amount of distilled water was added. Particular attention was paid to the absence from the test tube of visible impurities or air bubbles. The vitreous and the water were clearly distinguishable from each other in the test tube, the vitreous, being heavier, lay on the bottom. After these preparations, we placed a 4.0 Mhz searching unit with a diameter of five mm. against the surface of the water. With a view to eliminating disturbances and height-

ening sensitivity, the searching unit was plated with silver. We took photographs of the echoes which were traced on the scale of the Brown tube.

The vitreous in situ was studied as follows:

Shortly after slaughtering, a round hole, a window with a diameter of five mm., was made, exercising all care, in the sclera of the undamaged bovine eye, at first on the equator and later through the posterior wall in the vicinity of the papilla, the penetration of the hole being as far as the vitreous. A five-mm. searching unit with silver casing was then placed alternately against these windows, taking care not to break the vitreous, and photographs were made of the echo tracings visible on the scale. We then broke up the vitreous, by projecting through the window a four-mm. wide steel plate into the center of the vitreous, and turning it around a few times. The vitreous which had been broken in this manner was again examined with the searching unit through the windows in the sclera, and photographs were taken of the tracings obtained.

Results

1. The vitreous placed in the test tube rapidly developed a slight opacity which seemed to be composed of thin fibers. This opacity was best visible on the border between the water and the vitreous but it could also be seen deeper down within the vitreous.

Figure 2 illustrates the echoes obtained when the five-mm. searching unit was placed against the surface of the water. The area of the distilled water did not yield any echoes but the vitreous on the bottom of the tube emitted clear, low echoes. In our control investigations with a test tube entirely filled with water, we failed to elicit any echoes. The departure echoes seen on Figure 2 in the first part of the distilled water are due to the structure of the apparatus.

The echoes emitted by the vitreous were elicited best by studying them with the so-

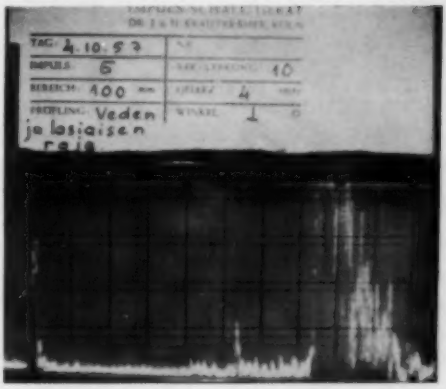

Fig. 2 (Oksala and Lehtinen). Illustrating the echoes obtained when the vitreous was placed in the test tube and distilled water was poured on it. Slight but clear echoes were obtained only from the area of vitreous lying at the bottom of the test tube.

called "depth magnifier," "Tiefenlupe" (fig. 3). In Figure 3, only the short remaining part of the distilled water is seen, which, as well as all the other part of it, did not emit echoes. Instead, a higher echo is clearly seen in the place marked by the arrow, the borderline between water and vitreous. The other echoes produced by the vitreous body are seen as numerous peaks of different height, whose position and height vary to a

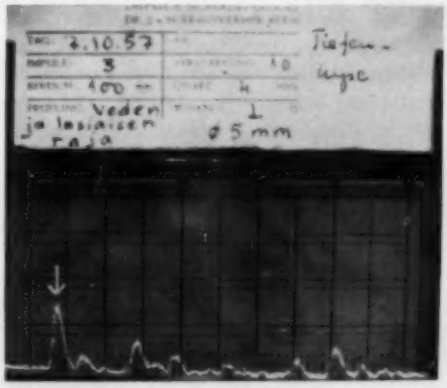

Fig. 3 (Oksala and Lehtinen). Echoes of the vitreous produced when examined by the "depth magnifier." On the border line between water and vitreous, marked by the arrow, is seen a higher than usual echo emitted by the vitreous.

Fig. 4 (Oksala and Lehtinen). The undamaged vitreous was examined in situ through a scleral window at the equator. The vitreous proved to be acoustically homogeneous.

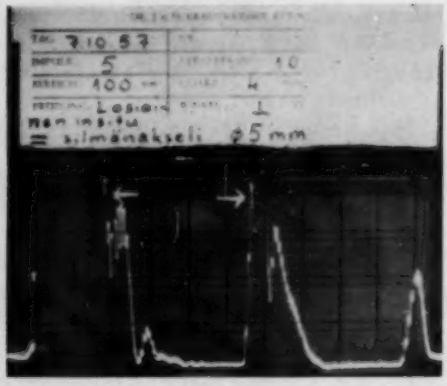

Fig. 5 (Oksala and Lehtinen). The undamaged vitreous has been studied through the scleral window in the vicinity of the papilla. Echoes could not be obtained from the vitreous area marked by arrows. The echoes seen in the first part of the vitreous are due to the searching unit itself.

restricted degree according to the movements of the searching unit and of the vitreous.

2. When examining the undamaged vitreous in situ through the scleral window situated on the equator, the echoes illustrated on Figure 4 were obtained; that is, only the departure echoes originated by the crystal and the echoes emitted by the opposite eye wall. The vitreous itself was not found to produce echoes.

When the searching unit was placed against the scleral window in the vicinity of the papilla, the echoes traced on Figure 5 were obtained. Since in this examination, performed almost in the axial direction, the eye became more compressed than in the former investigation, the area of research of the vitreous was much shorter.

In Figure 5, the vitreous is restricted to the area marked by the arrows. The departure echoes seen on these figures are again due to the apparatus itself. The echo representing the farther limit of the vitreous is generated on the border surface of the vitreous and the lens. Under ultrasonic examination, the undamaged vitreous was found to be acoustically homogeneous.

After we had broken the vitreous up in the manner already described by a thin metal plate, we conducted a new investigation with the ultrasonic apparatus, but this time only through the scleral window on the equator. Figure 6 shows the echoes then obtained by us. On Figure 6, slight but clear echoes could be distinguished in the middle of the vitreous and in its posterior part. The position and height of these echoes varied somewhat according to the movements of the crystal and of the vitreous. Sometimes these

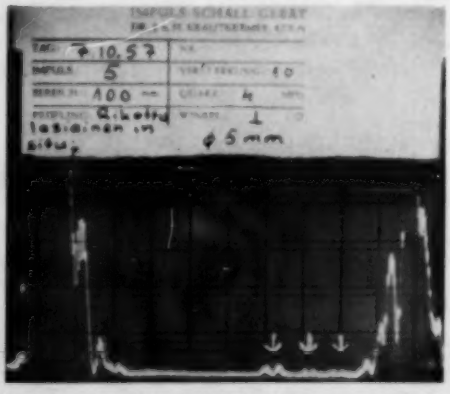

Fig. 6 (Oksala and Lehtinen). The mechanically broken vitreous has been examined in situ through the scleral window at the equator. In the central and end parts of the vitreous slight but clear echoes are seen at the places marked by the arrows.

echoes were as much as twice as high compared to those shown on Figure 6.

Discussion

In recent years have appeared several studies on the therapeutic use of ultrasound in eye diseases and on the changes which it brings about in various parts of the eye. On the other hand, the diagnostic application of ultrasound in ophthalmology has scarcely been studied at all.

Mundt and Hughes[10] found that use can be made of ultrasound in the diagnosis of tumors within the eye. Oksala and Lehtinen[11–13] have confirmed this and noted, moreover, that retinal detachment can be diagnosed by means of the echo emitted by the detached retina, also that its site and height can be determined even if the eye-grounds cannot be seen for some reason or other. These investigators have also found that ultrasound can detect and localize with an accuracy of approximately two or three mm. even small foreign bodies within the eye, whether it is possible to visualize them roentgenologically or not. Perhaps in the future we shall use parallel investigations by roentgen and ultrasound to locate foreign bodies inside the eye. On the basis of the studies of Oksala and Lehtinen it is further possible to carry out by means of ultrasound accurate measurements of the living eye and to diagnose rupture of the sclera even when it cannot be seen with the naked eye, slit-lamp, or through the ophthalmoscope.

In the study now presented we have for the first time used ultrasound in investigating the structure of the eye. The vitreous body, being optically and acoustically fairly homogeneous and moreover clearly delimited by its terminal surfaces, seemed to lend itself quite naturally as an object of research. The round shape of the eye also helped in the examination of the vitreous. The vastly different conceptions of the structure of the vitreous body still appearing in the literature proved a further incentive to this study.

The experimental results reported by us support in our opinion the point of view that the vitreous has its own fine micro- and macro-structure. An undamaged and healthy vitreous body is acoustically homogeneous to such an extent that echoes emitted by it cannot be traced by our ultrasonic equipment. But if we break up the vitreous, as we did in our experiments, either by pouring it into a test tube or by a mechanical process, the vitreous becomes acoustically much less homogenous and emits numerous echoes traced on the scale of the Brown tube.

Possibly by breaking the vitreous up in the manner described we produce in it an accumulation of thin fibers which are already capable of emitting echoes through the ultrasonic apparatus. This possibility may also be indicated by the observation that there is a constant variation in the localization and height of these small echoes on the scale. We have presumed these changes to be due to the circumstance that the ultrasonic waves touch upon these fiber accumulations from different directions, always according to the movements of the vitreous and of the searching unit.

These experimental observations might also have some clinical significance. It is known that several operators inject air or sodium chloride into the vitreous in connection with the operation for retinal detachment. If we accept the opinion that the vitreous has a very fine internal structure, damage to which easily leads to irreversible structural changes, the injection of air or sodium chloride would be a more or less serious trauma. Such a trauma could easily produce, by tearing of the fibers and their accumulation, opacities of the vitreous and disorders of its internal metabolism.

Summary

We have examined the acoustic properties of the vitreous in the bovine eye, as well as its structure, by means of an ultrasonic apparatus. The undamaged vitreous of the bovine eye proved to be acoustically homo-

geneous. When the vitreous was broken by two procedures—(1) by pouring it into a test tube and (2) by treating it mechanically with a metal plate—it was possible to obtain tracings on the scale of the Brown tube of numerous slight echoes from within the vitreous. We assume that these slight echoes originate as a result of the breaking up and accumulation of thin fibers in the vitreous body. By way of a clinical conclusion, we advance the opinion that injection of air or sodium chloride into the vitreous, sometimes practiced in the course of operations for retinal detachment, may be a serious trauma, leading to persistent opacity of the vitreous and producing disorders in its internal metabolism.

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A STUDY OF THE ELECTROCARDIOGRAPHIC ALTERATIONS*

Occurring during operations on the extraocular muscles

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The literature on ophthalmology frequently mentions cases of cardiac arrest and other myocardial disturbances during eye operations, particularly in those affecting the extraocular muscles. Not having observed similar accidents in our practice, we decided to perform a clinical and experimental study of the problem.

The physiopathologic explanation of these accidents is based on the action of the oculocardiac reflex induced by maneuvers on the extraocular muscles or by pressure on the eyeballs.

The oculocardiac reflex was first described in 1908 independently by Bernard Aschner[1] of Vienna and Giuseppe Dagnini[2] of Bologna.

The afferent pathway of the reflex consists in fibers which course with the ciliary nerves and the ophthalmic branch of the V nerve to the gasserian ganglion, then to the main sensory nucleus of the trigeminal in the floor of the IV ventricle; direct supranuclear fibers join this nucleus with the cerebral cortex passing through the lemniscus medialis. Stimulus reaches the nucleus of the X nerve and the efferent impulses are conveyed by fibers of the vagus to the depressor nerve which ends in the muscular tissue of the heart (fig. 1). The oculocardiac reflex is classically elicited by pressure on the eyeballs which rises excitatory impulses in the cardio-inhibitor center thus producing:

1. Slowness of the rate of the sinus rhythm (negative chronotropic effect).

2. Depression of the intracardiac conducting system (negative dromotropic effect).

3. Depression of the myocardiac contractility (negative inotropic effect).

As a consequence, bradycardia induced by the oculocardiac reflex might have its origin in:

1. Decrease of the ability of the sinus to stimulate impulses.

2. Idioventricular automatism created in an ectopic focus.

According to Sabena and Postelli[3] the following types of oculocardiac reflex may be distinguished:

1. Normal oculocardiac reflex: Reduction of eight to 10 beats per minute in the rhythm rate.

2. Positive oculocardiac reflex: Reduction of more than 10 beats per minute in the rhythm rate. This is the characteristic vagotonic response.

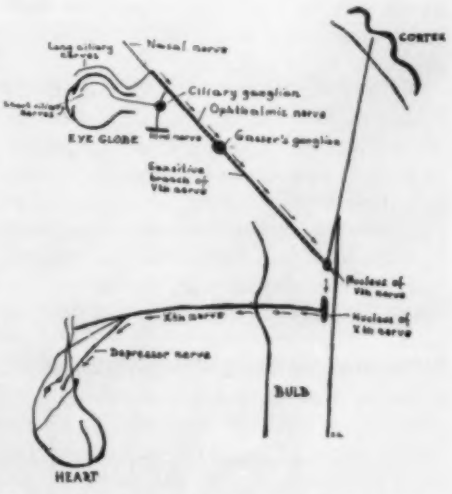

Fig. 1 (Rhode, et al.). Scheme of oculocardiac reflex. (Arrow) Reflex's pathways.

* From the Clinic of Ophthalmology, the Clinical Laboratory and Ophthalmic Research Department, Universidad Central de Venezuela, the Electrocardiographic and Cardiovascular Research Departments, National Center of Cardiology, and the Hospital Vargas. Presented at the IV Interim Congress of the Pan-American Association of Ophthalmology, New York, April, 1957.

3. Inverted oculocardiac reflex: Increasing of the heart rhythm. This is the characteristic sympatheticotonic response.

4. Absent oculocardiac reflex: It is seen in adults under normal physiologic conditions. As it is known, the oculocardiac reflex occurs less frequently in adults and it has been claimed by some authors that the reflex may be observed only in 50 percent of the subjects. Its occurrence decreases with age and becomes very rare in elderly people. In youngsters under 15 years of age the reflex is positive in about 90 percent of the cases.

There is a difference of opinion among authors regarding the time of compression required to elicit the reflex. Some of them believe that 10 seconds is enough, as stated in the classic technique of Dagnini, but others extend the compression for a period of five minutes. Nevertheless, it is generally accepted that the oculocardiac reflex is apparent after 15 to 20 seconds. Just the opposite is the observation of Merigot de Trigny[4] who claims that the reflex acts suddenly but becomes evident only after a latent period of two to three hours or less.

Without going into physiopathologic considerations about the oculocardiac reflex but taking only its cardiologic aspect, we shall make some observations on the subject as presented in the literature.

Merigot de Trigny[4] claims that general reaction manifestations accompanied by the oculocardiac reflex are seen very frequently in primary acute glaucoma. Sometimes these disturbances are the rule. The same author asserts that the reflex can be seen as a result of retrobulbar and subconjunctival hematomas, during enucleations, operations for squint, subconjunctival injections, and so forth.

Sorenson and Gilmore[6] mentioned recently a case of cardiac arrest in a girl, aged 13 years, as a result of manipulation of the medial rectus. They have auscultated the heart and controlled the pulse during operations for squint in 17 cases and they could observe bradycardia in 16 patients, seven of whom also had premature beats and one case suffered ventricular fibrillation. They concluded that such unexplained accidents occurring during operations for squint could be attributed to myocardiac disturbances due to impulses originated by excessive traction on the medial and lateral recti.

In their bibliography on cardiology Landman and Ehrenfeld[3] have published a case of ventricular fibrillation due to pressure on the eyeball.

As stated before, we have not observed in our practice any case of cardiac arrest during eye operations.

We have divided our work in two parts: (1) Clinical observations; (2) experiments on dogs.

1. Clinical observations

Material and Method

We have collected electrocardiograms in 13 patients, before, during, and after surgery, with a total of 14 operations (since one of the cases operated upon was for squint in both eyes). Ten of these cases were in males, three were in females. Two cases and three eyes were operated upon for squint by retroplacement of the medial recti and resection of the lateral recti. Eleven cases were enucleated for different diseases. The average age was 38.4 years, varying from 11 to 88 years. Seven patients were under 25 years of age and the other six were older. The electrocardiographic tracings were recorded at the following times:

1. *Preoperative.* (a) Before the anesthesia; (b) after the anesthesia.
2. When pulling the conjunctiva.
3. During the section of the conjunctiva.
4. When pulling each muscle.
5. During the section of each muscle.
6. During the section of the optic nerve in those cases requiring neurotomy.
7. During compression of the orbit, but only in cases of enucleation.
8. In the immediate postoperative period.

In 13 operations the electrocardiograms were recorded in Lead II. Lead III was used in one operation.

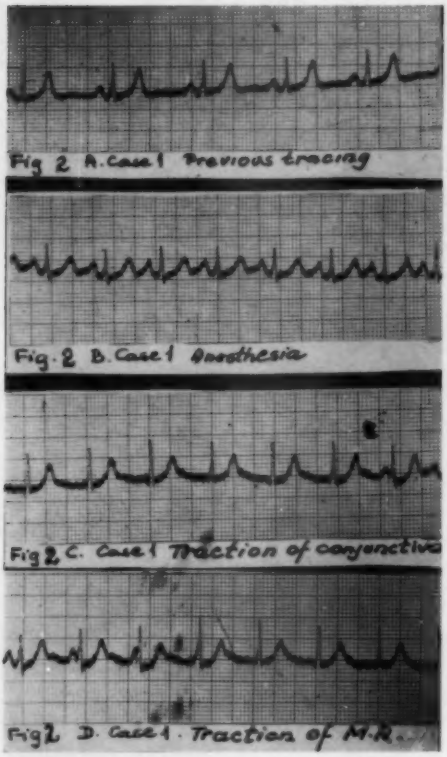

Fig. 2 (Rhode, et al.). Findings in Case 1.

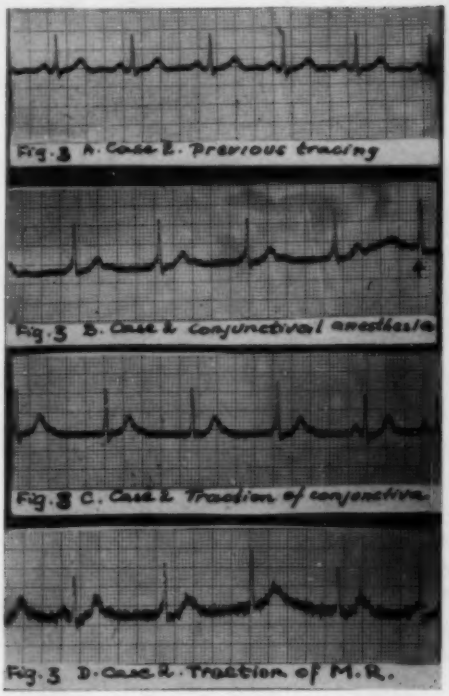

Fig. 3 (Rhode, et al.). Findings in Case 2.

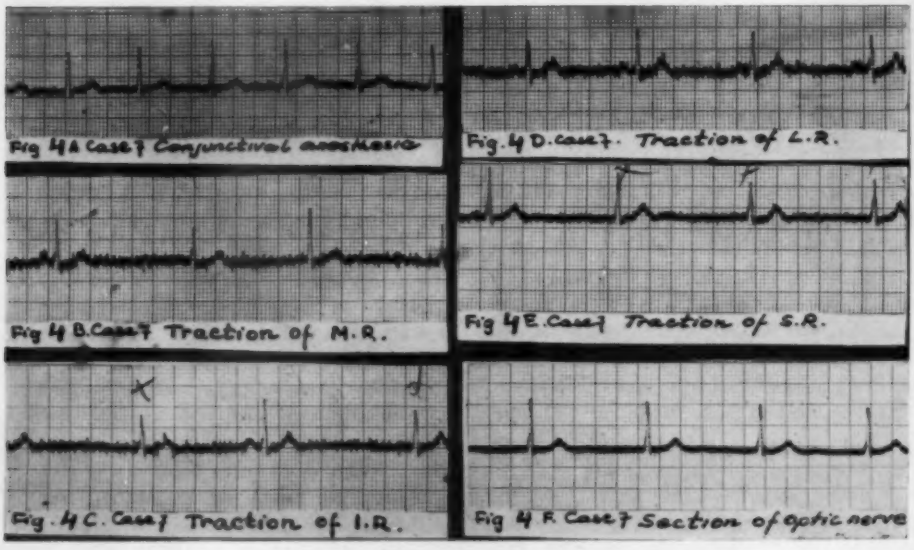

Fig. 4 (Rhode, et al.). Findings in Case 7.

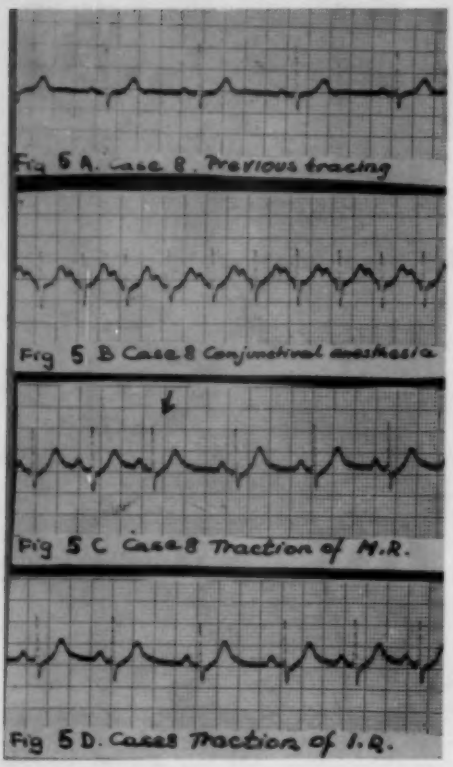

Fig. 5, Part I (Rhode, et al.). Findings in Case 8.

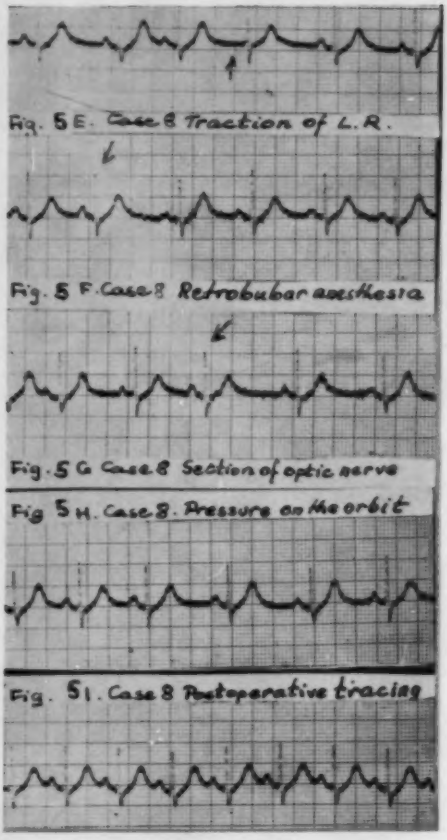

Fig. 5, Part II (Rhode, et al.). Further findings in Case 8.

Anesthesia was given as follows:

1. Topical two-percent cocaine solution and retrobulbar injection with five-percent Atoxicocaine-Adrenalina (six cases).

2. Topical two-percent cocaine solution and subconjunctival injection with five-percent Atoxicocaina-Adrenalina up to the ending of the muscular section, followed by retrobulbar injection with five-percent Atoxicocaina-Adrenalina given just before the neurotomy (one case).

3. Topical two-percent cocaine solution and subconjunctival injection with five-percent Atoxicocaina-Adrenalina up to the end of the muscular section, followed by general anesthesia with 2.5-percent pentothal (two cases).

4. General anesthesia with 2.5-percent pentothal (two cases).

5. General anesthesia induced with 2.5-percent pentothal, intubation and maintenance of the anesthesia with ether and nitrous oxide (two cases).

As preoperative medication, 25 mg. of Largactil were given intramuscularly to 11 patients the day before operation followed by an additional dose of 25 mg. the day of the operation. In two cases in which general anesthesia was administered, the preoperative medication was sodium seconal and atropine. Curare (Tecurin, three mg.-Anectina) was also given to these cases.

The results obtained are summarized in Table 1.

TABLE 1

SUMMARY OF CASES

Case #	Initials	Sex	History #	Diagnosis	Operation
1	T.B.	M	51084	Convergent strabismus, R.E.	O.D.: MR Retroplacement LR Resection O.S.: MR Retroplacement
2	E. P.	F	51186	Phthisis bulbi, L.E.	Enucleation, O.S.
3	P. P.	M	51376	Secondary absolute glaucoma, L.E.	Enucleation, O.S.
4	A. J.	F	172	Absolute glaucoma, O.D.	Enucleation, O.D.
5	R. P.	F	51514	Absolute glaucoma, O.S.	Enucleation, O.S.
6	I. R.	M	51529	Atrophia dolorosa, O.S.	Enucleation, O.S.
7	R. V.	M	51554	Secondary absolute glaucoma, O.S.	Enucleation, O.S.
8	F. G.	M	52881	Panophthalmitis, O.S.	Enucleation, O.S.
9	Ll. R.	M	52440	Phthisis bulbi, O.S.	Enucleation, O.S.
10	B. P.	M	52524	Absolute glaucoma, O.D.	Enucleation, O.D.
11	J. J.	M	52522	Panophthalmitis, O.S.	Enucleation, O.S.
12	O. R.	M	52802	Atrophia dolorosa, O.D.	Enucleation, O.D.
13	B. V.	M	52987	Concomitant strabismus, O.U.	Retroplacement medial rectus; resection lateral rectus

RESULTS

1. Tachycardia was present in nine cases when general or local anesthesia was administered (emotional).

2. Alterations of the rhythm: three cases treated with local anesthesia (disturbances of auriculoventricular conduction, nodal rhythm, and nodal escapes with auricular arrest). Two cases treated with general anesthesia (nodal rhythm).

3. Positive oculocardiac reflex: four cases treated with local anesthesia (total nine cases). Two cases treated with general anesthesia (total six cases). One case to be discussed: with positive oculocardiac reflex under local anesthesia the reflex disappeared when general anesthesia was applied.

4. No alterations: four cases treated with local anesthesia. Two cases treated with general anesthesia.

DISCUSSION

The results of our experiments show that

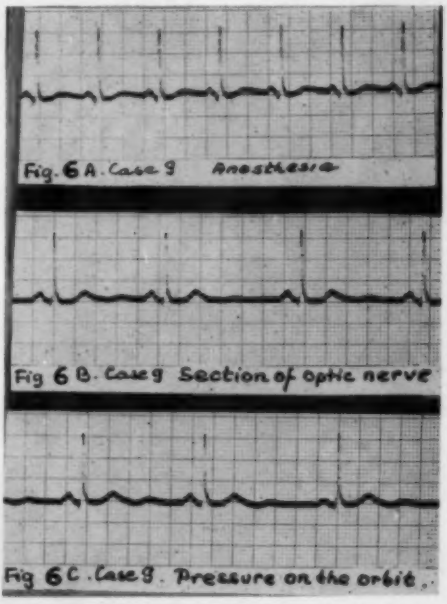

Fig. 6 (Rhode, et al.). Findings in Case 9.

TABLE 2

ELECTROCARDIOGRAPHIC STUDY

Case #	Age (yr.)	Anesthesia	Electrocardiograms
1	20	Topic 2% cocaine solution. Retrobulbar with 5% atoxicocaina-adrenalina	a. Control tracing: shows marked vagotony b. Under local anesthesia the rhythm rate increases from 68 to 113 beats per minute c. When pulling the conjunctiva the rhythm remains in about 100 beats per minute; P wave is absent (possible auricular arrest). The last fast complex is preceded by a P wave with normal characteristics but P-R segment shortens d. Shows the changes induced by the traction of rectus muscle. It may be seen that the P-R segment shortens in the #1 and 2 complexes up to its fusion with QRS thus slurring the R upstroke (#3 and 4 complexes) similarly as it is observed in Wolf-Parkinson-White cases.—P wave is absent in #5, 6 and 7 complexes. The rhythm rate is not modified (fig. 2)
2	88	Topic 2% cocaine solution. Retrobulbar with 5% atoxicocaina-adrenalina	"A" is the control tracing in Lead I. When the conjunctival anesthesia is injected a Wolf-Parkinson-White becomes present and the rhythm rate decreases from 75 to 63 beats per minute. The last fast complex, which is marked with an arrow, has the same characteristics as the control tracing and is not preceded by P wave. "C" and "D" were recorded during traction of the conjunctivas and do not show any changes in reference to B; some complexes simulate the morphology of WPW; in others there is the shortening of P-R (latest two); the former ones show P wave absent (fig. 3)
3	23	Topic 2% cocaine solution. Retrobulbar with 5% atoxicocaina-adrenalina	No alterations
4	63	Topic 2% cocaine solution. Retrobulbar with 5% atoxicocaina-adrenalina	Tachycardia
5	80	Topic 2% cocaine solution. Retrobulbar with 5% atoxicocaina-adrenalina	Tachycardia
6	16	Topic 2% cocaine solution. Subconjunctival injection with 5% atoxicocaina-adrenalina. No retrobulbar injection but general anesthesia with pentothal just before neurotomy	Control tracing shows tachycardia. Bradycardia was developed at the time of every operative step. Pressure on the eyeball or on the orbital vertex did not produce any modification
7	39	Topic 2% cocaine solution. Subconjunctival injection with 5% atoxicocaina-adrenalina. No retrobulbar injection but general anesthesia with 2.5% pentothal just before neurotomy	Bradycardia was developed when pulling every muscle. Slight Bradycardia appeared by compression of the orbital vertex. Nodal rhythm (fig. 4)
8	20	Topic 2% cocaine solution. Subconjunctival injection with 5% atoxicocaina-adrenalina. After section of all muscles retrobulbar injection with 5% atoxicocaina-adrenaline was given	a. Control tracing. b. Subconjunctival anesthesia: tachycardia of 150 beats per minute is developed. c and d. Bradycardia is developed when pulling the recti muscles. In "E" a nodal escape is marked with an arrow. f. Retrobulbar anesthesia. g and h. These tracings were successively recorded during section of the optic nerve and pressure on the orbit; a moderate bradycardia may be appreciated (fig. 5)
9	48	General anesthesia with 2.5% pentothal	Tachycardia. Positive oculocardiac reflex (fig. 6)

TABLE 2 (*Continued*)

Case #	Age (yr.)	Anesthesia	Electrocardiograms
10	63	Topic with 2% cocaine solution. Retrobulbar injection with 5% atoxicocaina-adrenalina	a. Control tracing. b. After the anesthesia and during the pull of the SR there are Bradycardia and a nodal escape or lower auricular (second tracing) c. Orbit compression elicit bigeminy rhythm caused by auricular premature beats (fig. 7)
11	11	General anesthesia with 2% pentothal	Tachycardia. Symptoms of subendocardial lesion of the left ventricle appeared. Traction on the ocular muscles the same as orbital pressure ("C," "E," "D") developed inferior nodal rhythm and sinoauricular block with nodal escape. (Arrow in "D," fig. 8)
12	16	Induction with 2.5% pentothal. Injection of 3 mg. tecurin. Intubation and maintenance of general anesthesia with ether and nitrous oxide	Any changes were elicited by the different ocular maneuvers (fig. 9)
13	14	Induction with 2.5% pentothal. Injection of Anectina. Intubation and maintenance of general anesthesia with ether and nitrous oxide	Tracing "B" was recorded during traction of the Recti muscles; it shows absence of P wave and slowness of the rhythm from 125 to 100 beats per minute (fig. 10)

TABLE 3

RESULTS

Case #	Local Anesthesia	General Anesthesia
1	Tachycardia. Alterations of PR	
2	No alterations	
3	Tachycardia. No alterations of electrocardiogram deflections	
4	Tachycardia. No alterations of electrocardiogram deflections	
5	Tachycardia. No alterations of electrocardiogram deflections	
6	Tachycardia. Positive oculocardiac reflex	Negative oculocardiac reflex
7	Positive oculocardiac reflex. Disturbances of rhythm (nodal rhythm)	Disturbances of rhythm (nodal rhythm)
8	Tachycardia. Positive oculocardiac reflex	
9		Tachycardia. Positive oculocardiac reflex
10	Positive oculocardiac reflex. Disturbances of rhythm (nodal escape)	
11		Tachycardia. Disturbances of rhythm (nodal rhythm)
12		No alterations
13		Tachycardia. Positive oculocardiac reflex

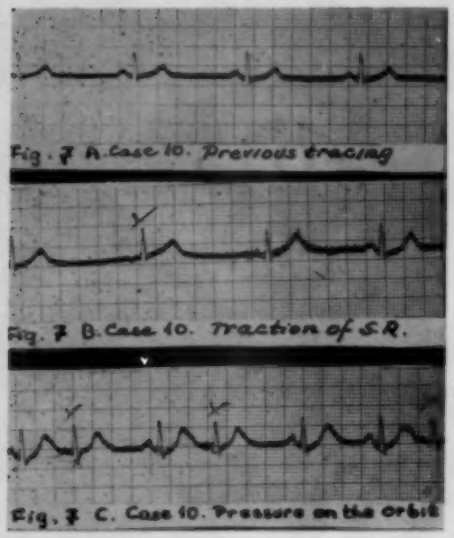

Fig. 7 (Rhode, et al.). Findings in Case 10.

tachycardia was present in 61 percent of the cases. It developed in all cases during the application of general or local anesthesia. This fact leads us to think that emotional factors are responsible for its production.

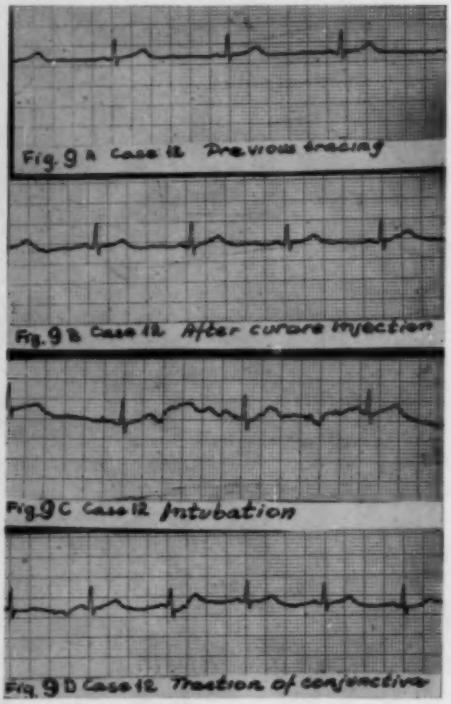

Fig. 9, Part I (Rhode, et al.). Findings in Case 12.

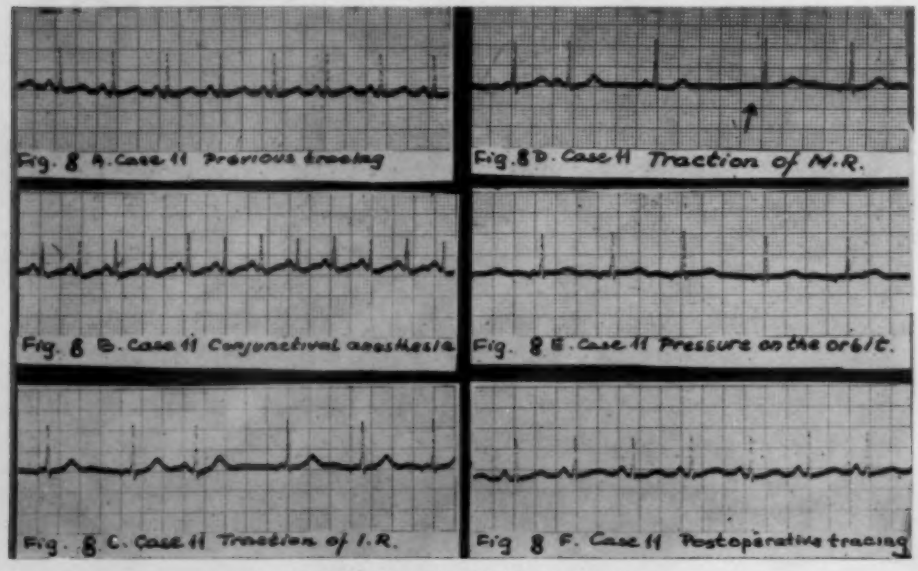

Fig. 8 (Rhode, et al.). Findings in Case 11.

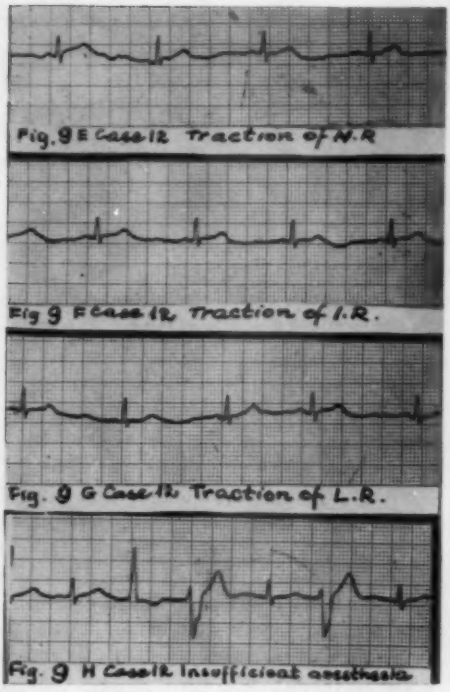

Fig. 9, Part II (Rhode, et al.). Further findings in Case 12.

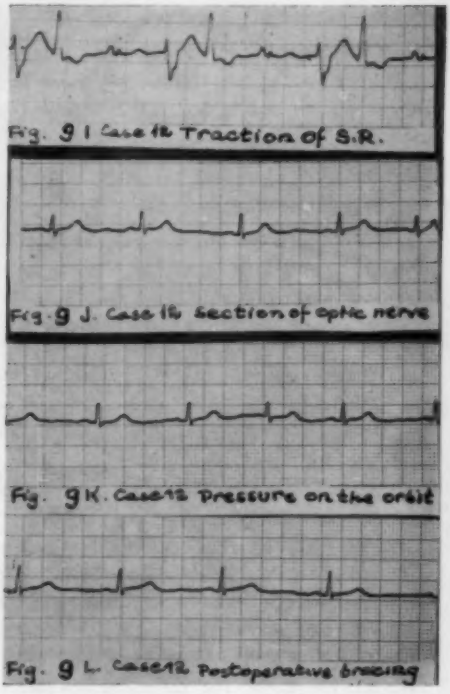

Fig. 9, Part III (Rhode, et al.). Further findings in Case 12.

In 44.44 percent of the cases treated with local anesthesia, the different operative maneuvers did not produce any change, while with general anesthesia there were no changes in 33.33 percent of the cases.

The oculocardiac reflex was positive in 44 percent of the cases treated with local anesthesia as well as in 33 percent of the cases in which general anesthesia was applied.

It should be noted with regard to the oculocardiac reflex that one case registered positive when local anesthesia was applied but disappeared as soon as general anesthesia was given.

Rhythm disturbances were observed in five cases of the whole group and the percentage (33 percent) was the same for cases treated with local or general anesthesia.

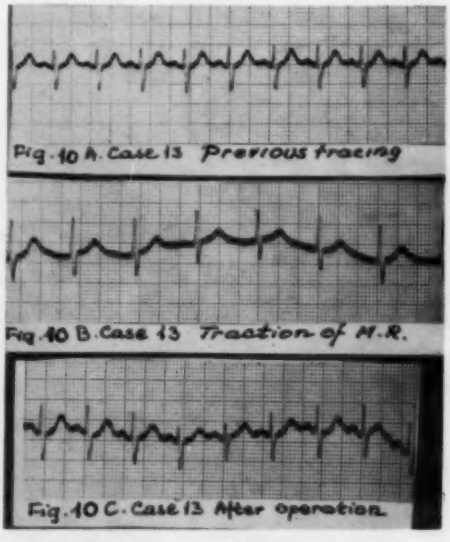

Fig. 10 (Rhode, et al.). Findings in Case 13.

2. Experiments on dogs

The electrocardiographic changes found in the human as a result of maneuvers on the eyeballs led us to conduct an experimental study in an effort to determine the etiologic factors.

Material and Method

A group of 10 young dogs was used; each of them 6.0 to 8.0 kg. in weight. Preliminary electrocardiograms on all of the dogs showed no myocardial damage in any of them.

The anesthesia administered in several cases was intraperitoneal Nembutal at the rate of 420 mg. per kg.; in other cases we used 2.5-percent solution of sodium pentobarbital given intravenously at varying doses from the useful one to the lethal one able to produce respiratory arrest.

Investigation of the oculocardiac reflex was made before and after anesthesia with the animal in a normal condition as well as after promoting anoxemia for variable periods of time.

In all the cases the different operative steps of the enucleations were performed to make more evident any changes that might be caused.

The recording instrument used was a Schwarzer E-502 electroencephalograph of six channels and direct inscription at variable speeds from 30 to 60 mm. per second. In all the cases we recorded the standard leads and also the unipolar of the fore right leg in order to have good control of the respiration. In each case, artificial respiration was applied with a Harvard pump through tracheal intubation. The results of our experiment were uniform.

Results

In the majority of our experiments, the pressure on the eyeballs in the conscious animal developed a marked bradycardia of sinusal type; there was not at any moment the slightest evidence of alterations in the different parts composing the electrocardiogram. The experiment shown in Figure 11 illustrates this situation. Lead II in control conditions may be seen in the upper tracing presenting a sinusal arrythmia of the vagal type which becomes more accentuated when the eyeballs are compressed (lower tracing); in addition, the intervals R-R elongate. Segments PR, ST, and QT, the same as P and T waves, do not show any changes.

When the animal is anesthetized there is no bradycardia as a result of compression on the eyeballs and the cardiac rate remains uniform and indifferent to the ocular maneuvers. The QRS complex, the P and T waves, and the PR, ST, and QT segments

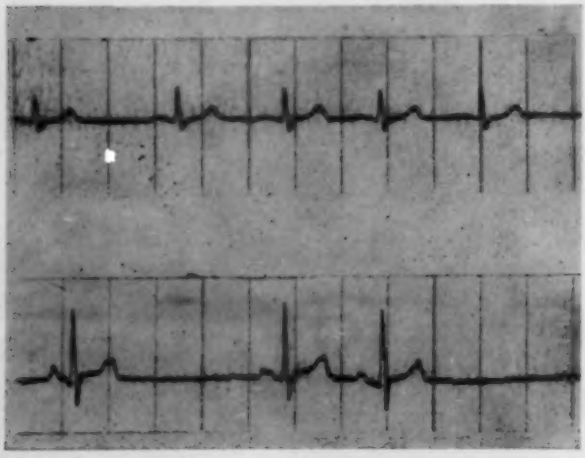

Fig. 11 (Rhode, et al). Animal experiment. Pressure on the eyeballs of a conscious animal produced marked bradycardia.

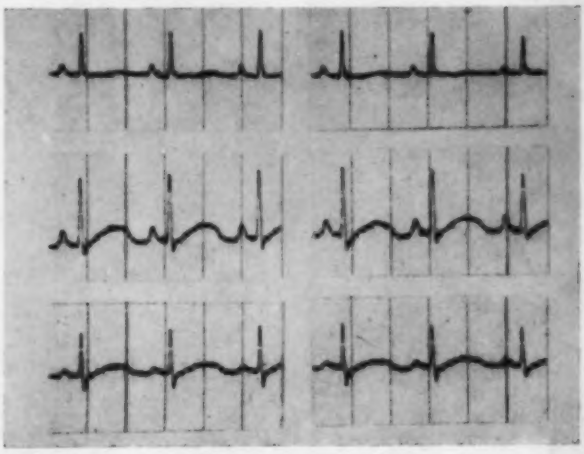

Fig. 12 (Rhode, et al.). Animal experiment in which both eyes were enucleated. See text for explanation.

do not show any changes. The different operative steps for the enucleation of the eyeballs on the anesthetized dog did not produce any modification of the electrocardiogram.

Figure 12 demonstrates an experiment in which both eyeballs were enucleated. Column 1 shows Lead I (upper tracing), Lead II (middle tracing), and Lead III (lower tracing) being recorded under control conditions, with the dog anesthetized and receiving artificial respiration. Column 2 corresponds to the same leads when a strong pressure is exerted on the eyeballs. It can be seen that there are neither changes in rhythm nor in the components of the electrocardiogram. Figure 13 corresponds to the same experiment and shows the standard leads during the final steps of the operation. Again, the electrocardiogram does not show any modification.

With the purpose of studying the depth of anesthesia required to prevent the oculocardiac reflex, several experiments were made giving variable but insufficient doses of pentothal. In most of the cases, a slight bradycardia developed but in others the beat rhythm remained unchanged. This is illustrated in Figure 14 where Lead I is the upper tracing, Lead II is the second tracing, Lead III is the third tracing, and VR is the lower tracing. In this case, the dog received only hypnotic doses of the anesthetic. Column 1 represents the control conditions and column 2 the recording obtained when both eyeballs were compressed. The comparative study of both tracings showed no modification.

In order to study the electrocardiographic

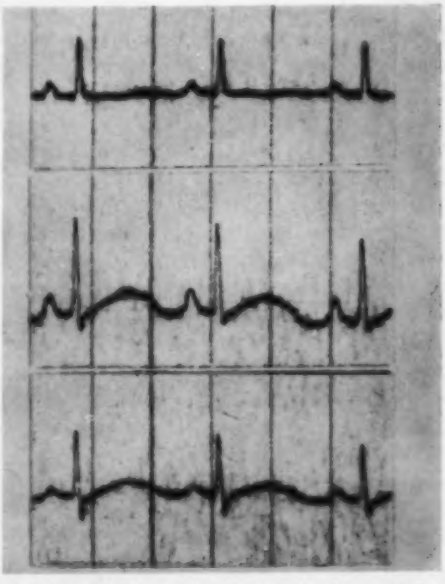

Fig. 13 (Rhode, et al.). Same animal experiment as in Figure 12. See text for explanation.

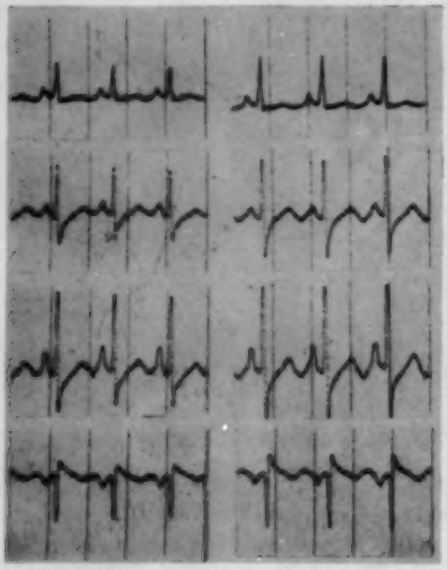

Fig. 14 (Rhode, et al.). Animal experiment in which pentothal was administered. See text for explanation.

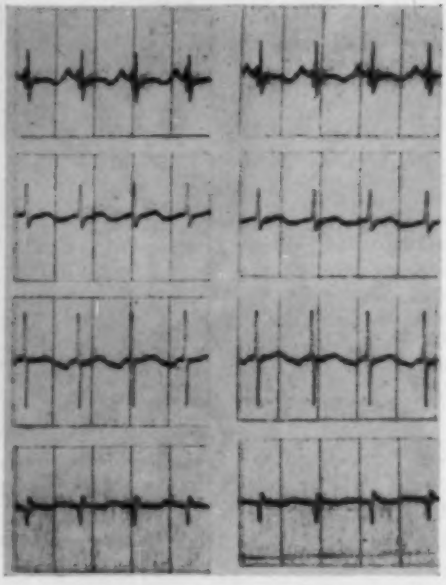

Fig. 15 (Rhode, et al.). Results of animal experiments. See text for explanation.

changes produced by the ocular maneuvers in conditions of anoxemia, we proceeded in the following manner: anesthetized the dog with a useful dose of pentothal; recorded control tracings; and immediately set up tracheal intubation. Respiratory arrest was then produced with toxic doses of the same anesthetic and followed by artificial respiration.

New electrocardiographic control tracings in the various leads showed no alterations. Under these experimental conditions different degrees of anoxemia may be obtained by disconnecting the pump which supplies the artificial respiration. In this anoxemic situation we have obtained various types of arrythmia, modifications of ST segment and P waves, thus giving evidence of different degrees of ischemia. Under these conditions neither surgical maneuvers on the eyeballs nor pressure on them or on the orbit produced electrocardiographic modifications in the experimental animals.

The results of these experiments are presented in Figure 15 to Figure 20 which show Lead I (first tracing), Lead II (second tracing), Lead III (third tracing), and VR (fourth tracing); column 1 corresponds to

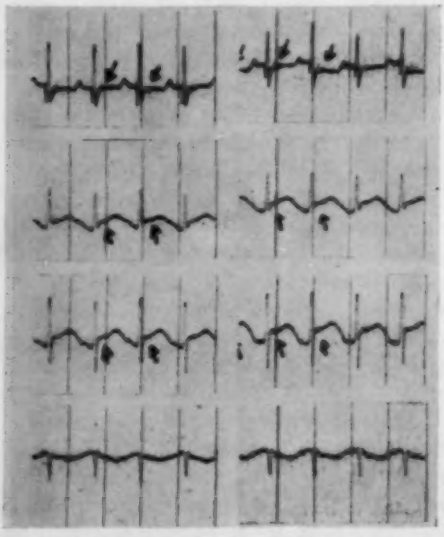

Fig. 16 (Rhode, et al.). Results in animal experiments. See text for explanation.

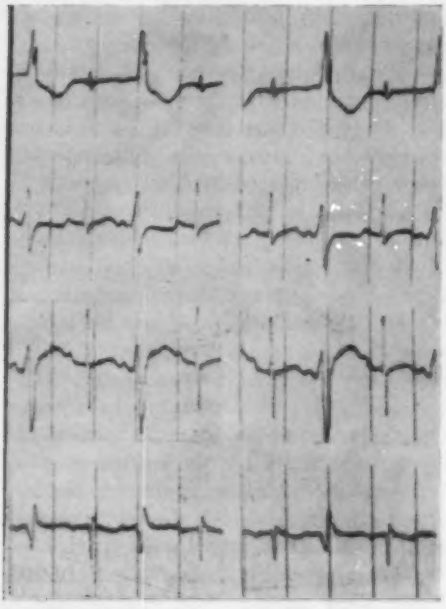

Fig. 17 (Rhode, et al.). Results in animal experiments. See text for explanation.

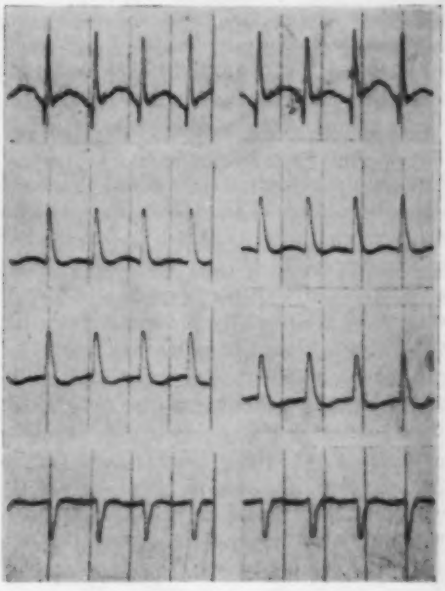

Fig. 19 (Rhode, et al.). Results in animal experiments. See text for explanation.

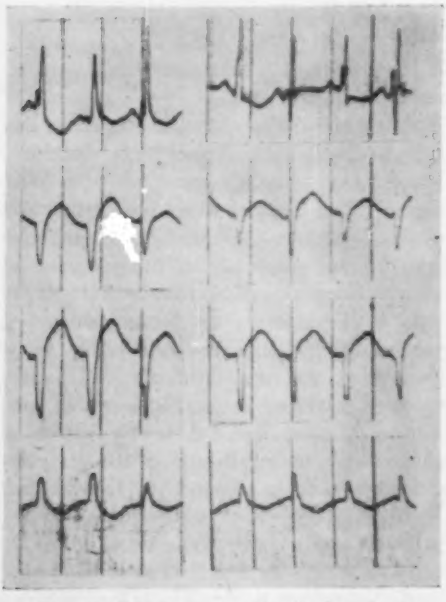

Fig. 18 (Rhode, et al.). Results in animal experiments. See text for explanation.

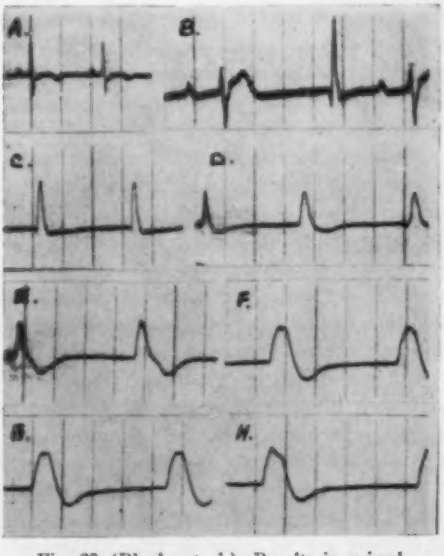

Fig. 20 (Rhode, et al.). Results in animal experiments. See text for explanation.

control conditions and column 2 shows the result of the pressure on the eyeballs. Column 1 in Figures 16, 17, 18, and 19 demonstrates the different degrees of anoxemia produced in the animal. After two and one-half minutes of anoxemia, the segment ST in Lead I appears negatively displaced and the same segment in Leads II and III shows a positive displacement (fig. 16). After three and one-half minutes (fig. 17) alternate rhythm may be appreciated with the normally conducted complexes followed by the morphology of right bundle-branch blocking. The normally conducted QRS complexes have flatten T and those corresponding to the right BBB show marked displacements of ST, negative in Lead I and positive in Lead III.

After five minutes of anoxemia the P waves disappear and morphology of right BBB may be observed (fig. 18). Finally, after six minutes of anoxemia the type of disturbance in the conduction system is inverted thus appearing left BBB (fig. 19) and marked displacements of ST segment, positive in Lead I and negative in Lead III.

In these conditions, artificial respiration was given again, thus producing the gradual disappearance of modifications and return to the control conditions. Being acquainted with the alterations which anoxemia may produce on the heart and knowing the resulting changes in the electrocardiograms, we again reproduced the condition of anoxemia by interrupting the artificial respiration and then proceeded with the surgery and the pressure on the eyeballs. In these conditions we observed that the ocular maneuvers did not introduce any electrocardiographic changes in addition to those previously obtained under the conditions of anoxemia.

Column 2 in Figures 16, 17, 18, and 19 represent different operative steps and different moments of pressure during the various degrees of anoxemia already mentioned (column 1). Aside from the alterations for which the anoxemia is responsible, it appears that there are no other electrocardiographic changes.

In Figure 20 another experiment is presented in which anoxemia was promoted during six minutes. Tracing "A" shows Lead I under control conditions. In "B" a disturbance of the auriculoventricular conduction with the type of first degree A-V blocking is apparent; also sinoauricular blocking with nodal escape and positive displacement of ST segment. In "C" auricular arrest with nodal rhythm and conduction disturbances of the left bundle-branch may be observed. Between the first and second complexes of tracing "D" an increase of the block (the same as in between the complexes 1 and 2 of tracing E) may be observed. "F," "G," and "H" point out more advanced degrees of the conduction disturbances. In this experiment, the restitution of the artificial respiration made the modifications return to the conditions of control. When the anoxemia was again established, the electrocardiographic changes reappeared in the same sequence. Under these conditions the surgical maneuvers and pressure on the eyeball did not induce modifications other than those attributable to the anoxemia.

Discussion

The results of our experiments point out that, in a high percentage of cases, pressure on the eyeballs in the conscious dog elicits the oculocardiac reflex evidenced by bradycardia. These facts are in agreement with the findings of those authors who have studied the subject.[8] In one of our experiments, the pressure on the eyeballs accentuated a previously existing sinusal arrythmia of the vagal type. In a few cases, the ocular maneuvers produced tachycardia; this fact has been explained by some as a consequence of the excitement produced in the dog by those maneuvers.[8] In any one of these cases we could observe the modifications of the P wave claimed by some as a result of the tachycardia. Prolongation of the interval PR and the depression and negative deflections of the P wave as previously described with the bradycardia[8] were not observed in any of our experiments.

Inhibition of the oculocardiac reflex was

always produced by anesthesia. Ocular maneuvers performed during deep anesthesia did not modify the rhythm of the cardiac beat. No other modification of the different parts of the electrocardiogram under the same conditions was recorded. The results of our experiments showed no changes of P and T waves including the fast complex or the segments PR, ST, and QT when pressure on the eyeballs was exerted or during the surgical maneuvers for the enucleations.

These results are contrary to the findings of other authors[8-13] who have described modifications of T wave and the segment ST-T in most of their anesthetized animals.

The cases with conditions of anoxemia we have studied have constantly shown as the first electrocardiographic modification: disturbances of ventricular repolarization with the type of myocardiac ischemia made evident by modifications of T wave; zones of injury printed out by positive or negative displacements of the segment ST. These disturbances are reversible since they disappear when artificial respiration is given. This fact makes evident the anoxemic origin of the disturbance.

These findings have been observed before in cases of severe barbituric intoxications[14] and their prevention by means of artificial respiration has been recommended.[15] It has been claimed by some that this fact is partially due to cardiac anoxemia caused by the depression of the respiratory center, the sympathetic stimulation and the tachycardia developed by hypotension.

The results of our experiments do not agree with these assertions. We consider that anoxemia is the only factor responsible for this alteration, since its reversal is obtained solely by pulmonary hyperventilation, not changing during the experiment the other factors (rhythm, tension, and so forth).

Being familiar with the modifications that anoxemia is able to produce and having obtained in our experiments no modification of the electrocardiogram during the manipulations on the eyeballs of anesthetized animals, we suggest that the modifications described by some could be due to anoxemia caused by depression of the respiratory center, attributable to the reflex effect induced by vagal stimulation. By prolonging the period of anoxemia, it was possible to obtain first-degree auriculoventricular block, sinoauricular block, alternating rhythm, auricular arrest, increase in the ventricular automatism, idioventricular and nodal rhythms with bundle-branch block. Most of these alterations were reversed when respiration was re-established artificially. When these disturbances were reproduced in the same experiment, the compression and surgical maneuvers on the eyeballs did not elicit modifications other than those previously produced by anoxemia.

These results lead us to believe that the oculocardiac reflex is interrupted in the anesthetized animals, only if the myocardic conditions are altered by anoxemia in any one of its phases.

We have to state that the vagal stimulation in all our experiments was made by exerting pressure on the eyeballs and by pulling the extraocular muscles; it must be further demonstrated whether a more intense vagal stimulation (electric, chemical, and so forth) would be able to produce modifications in the electrocardiogram under the same experimental conditions. This subject will be studied in future experiments.

Conclusions

Our clinical observations and experimental work on dogs lead us to conclude:

1. Surgical maneuvers on the extraocular muscles are not sufficient by themselves to produce deep alterations in the physiopathology of the heart and are certainly unable to cause cardiac arrest.

2. We believe that anoxemia is responsible for the alterations of the electrocardiogram found by us in experimental animals.

3. Hyperventilation normalizes the altered heart rhythm and prevents appearance of the oculocardiac reflex.

4. Cardiac arrest observed by other authors as a result of operative maneuvers on the extraocular muscles under general anes-

thesia, which has been attributed to the action of the oculocardiac reflex, is due, in our judgment, to accidents of anesthesia (anoxia).

5. With a good retrobulbar or general anesthesia we have not seen cardiac arrest during operations on the extraocular muscles or when pressure is exerted on the vertex of the orbit.

University Eye Clinic.

We must explain that all maneuvers on the extraocular muscles and pressures on the orbital vertex were made with unusual intensity with the purpose of developing those disturbances attributed to the oculocardiac reflex.

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CONGENITAL OCULAR MOTOR APRAXIA

Report of a Case in an Adult Male

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In 1952, Cogan (the Jackson Memorial Lecture) presented four young boys with a congenital clinical entity in which there was an inability of voluntary eye movements in the horizontal plane combined with compensatory jerky head movements which made up partially for the oculomotor deficiency.

These boys were unable to turn their eyes in response to a command or to look at an object that suddenly attracted their attention, or to move their eyes in response to a conscious effort without any apparent external stimulus. Reactions on optokinetic stimuli were largely disturbed in the horizontal plane. In the vertical plane the reactions were present. Except in one boy, who had an extrapyramidal disturbance, the other boys had no other neurologic anomalies.

In 1955, A. Urrets-Zavalía, Jr., and C. Remonda presented a fifth case in a six-year-old boy, who had an asymmetric disturbance. They made a table of the symptoms and signs as recorded in the five known cases. Both authors wondered whether the condition had been, so far, observed only in boys and at an early age.

In 1954, my father presented before the Dutch Ophthalmological Society a case of congenital ocular motor apraxia in an adult male. Owing to the death of my father soon after, this case was never published. Still one can wonder whether the condition has never been observed in females.

Case report

The patient whom we saw first in 1953, was then aged 51 years. He was youngest of nine children, two of whom had died at an early age and one later. His parents had normal eyes and, as far as is known, normal eye movements. His father had a job in the Dutch railways and so had his brothers. His brothers and sisters also had normal eyes and normal eye movements. The patient had always been healthy. He had been squinting since birth. At the age of six years he was operated on for squint (tenotomy of both internal rectus muscles). Except squinting, no other eye anomalies were noticed at that time. However, according to his older sister, he had had typical jerky head movements since birth. At school he was an average pupil.

In 1926 (at the age of 24 years), his rare anomaly was first discovered by his ophthalmologist (Dr. Pieltjes), whom he consulted for reading difficulties.

The patient has been working at a cork factory for many years. He has reading glasses which he does not wear at his work in the factory. With glasses he reads the usual newspaper prints. He is married and has two children, both without any eye trouble.

It is impossible for the patient to direct his eyes voluntarily to a certain object in the horizontal plane. To get adjusted to a certain object he makes a jerky head movement, mostly to the right side, sometimes to the left side, but considerably farther than the object of attention, producing thus a characteristic overshoot. Once the eyes have fixed the object, the head returns slowly to the direction of the object of fixation. There is a continuous tendency to a "déviation conjuguée" to the left. This appears either involuntary, or by trying to adjust, or by lid winks. Involuntary eye movements are possible in all directions. They are conjugated and mostly submaximal. Eye movements to the right are restricted to two thirds of the normal field of gaze. Mostly the eyes turn to the left. The patient does not feel these movements, he only becomes aware of the changing scenery of his surroundings. Following movements are absent in the horizontal plane, present in the vertical plane.

Once fixating, the patient is able to hold fixation for some time while his head is turned slowly, actively or passively to either side in the horizontal plane. Nevertheless there is always the tendency of the eyes to deviate to the left. If the head is turned quickly, there appears a compensatory reaction of the eyes, as clearly as in newborn babies. Turning the head in the vertical plane creates similar reactions.

There is an alternating divergent strabismus of 10 to 12 degrees. Cover-test reactions cannot be evoked. Convergence reactions are impossible. Pupillary reactions and visual fields are normal.

Vision is: O.D., 6/15, with a +1.0D. sph., 6/15; O.S. 6/30, with a +1.0D. sph., 6/20. Both fundi are normal.

To get an idea as to the cause of the condition the patient was investigated on: (A) the optomotor reactions; (B) the vestibular reactions; (C) electro-encephalography; (D) the head movements shown by means of slow-motion moving pictures; (E) further possible neurologic anomalies.

A. Optomotor reactions

There is no nystagmus, either in the primary position or when the eyes are directed to the right or to the left side. Lid winking and conscious fixation sometimes provoke a "déviation conjuguée" to the left. If this is considered to be the slow phase of a nystagmus latens, a quick phase is not present and cover reactions cannot be evoked. There is no ability for adjusted movements in the horizontal plane, either involuntary or voluntary. Following movements are inperceptible or absent.

Optokinetic stimuli, both eyes, increased the gaze tonus to the left. Optokinetic nystagmus showed, binocular, with movement to right, jerky nystagmus to the left; binocular, with movement to the left, deviation of both eyes to the left. Monocular, R.E., with movement to the right, first eyes at rest, afterward an inverse type of nystagmus; monocular, R.E., with movement to the left, single jerky movement to the right; monocular, L.E., with movement to the right, slow jerky movements to the left of a larger amplitude; monocular, L.E., with movement to the left, immobility; sometimes deviation of both eyes to the left.

The results show that the optical gaze tonus is low and that the fixation reflexes did not develop in a normal way.

B. Vestibular reactions

Compensatory reactions can easily be evoked in this patient, in contrast to normal adults, possibly due to an insufficient inhibition by the optical reflexes. Caloric nystagmus and rotating nystagmus are normal.

C. Electro-encephalogram

The electro-encephalogram (occipital pole) is somewhat depressed.

D. Head movements by slow-motion pictures

It is clear that the patient tries to compensate for the failure of his optical adjustment reactions by means of his head movements and the vestibular (compensatory) reactions. Thus he returns to a lower phylogenetic level, where the position of the eyes is chiefly ruled by vestibular stimuli. By jerky horizontal head movements in the direction of the intended object, the eyes are moved in the opposite direction and subsequently are carried along in this maximal lateral position until one of the eyes is directed to the intended object. Once fixating, the head is now returned slowly a certain amount of degrees to place the eye in the primary position.

In this patient, the head turning evokes two vestibular reflexes: The first, by the jerky head movement, results in a deviation of both eyes in the opposite direction; afterward, by a slow head movement, the vestibular stimuli help the poor fixation reflexes get the eyes in the primary eye position without losing the fixation. The film (for which we thank Prof. H. M. Dekking) illustrates these typical head movements clearly.

Discussion

Reviewing the results of this investigation, the following conclusions can be made:

1. There are no reflectory optical adjustment reactions in the horizontal plane.

2. No voluntary eye movements for adjustment can be performed in the horizontal plane.

3. Only poor fixation reflexes are present in the horizontal plane, as shown by maintaining of fixation, once the eyes have fixated an object, when the head is turned slowly, actively or passively. These fixation reflexes can easily be disturbed by eyelid winking or by the tendency to a "déviation conjuguée.

4. There is a disturbance in the development of the monocular optomotor reflexes, as shown by the former convergent strabismus and the low visual acuity.

5. There is also a disturbance in the development of the conjugated optomotor reflexes, as shown in the weak fixation reflexes and the low gaze tonus.

6. For the optical adjusting movements is substituted a reaction evoked with the aid of head movements and vestibular stimuli.

7. There is a dominance of compensatory reactions if the patient turns his head quickly in the horizontal plane.

If we now assume "voluntary" adjusting movement of the eye as a reaction superimposed on the adjusting reflex with the aid of associated functions, it is clear that this higher reaction could not develop when the lower one failed. In this way the second reaction then becomes a result of the first. In our case, therefore, it is not necessary to claim that failure of voluntary adjusting movements is a result of a disturbance in the frontal cortex, the diencephalon, or in the brain stem.

The ocular symptoms of this patient resemble the eye movements of a rabbit—in the rabbit the eyes cannot move voluntarily but the head can. Optokinetic nystagmus can be evoked in the absence of nonmoving contours. So this nystagmus is weaker, as in man. When contours are moved from the back toward the front (noseward in man), there is a stronger reaction than when objects are moved from the front to the back (templeward).

But why are there no voluntary eye movements? We can consider two possibilities:

1. The optomotor reflexes were not normally joined with the subcortical centers for eye movements but were attached to the pathways for head movements.

To illustrate this condition, we present the "innervation tonus" theory by Otto Roelofs. In this theory the position of the eyes

is controlled by the stimuli of the centers of co-ordination—named by Roelofs the "gaze tonus." This gaze tonus is composed of a nonoptical and an optical component. Before birth the "tonizing" of the centers of co-ordination (centers of gaze) is induced by nonoptical stimuli. The "grafting" of cortical stimuli on the subcortical pathways, coming from these centers, can now occur because, in these centers for co-ordination, the optical stimuli gradually dominate the nonoptical stimuli, thanks to the development of the optical cortex. The "restraining and controlling task of the cortex" on subcortical reactions can be explained in this way.

Returning to our patient we see that in him the optical component of the gaze tonus is so underdeveloped that the nonoptical component has remained dominant. The low gaze tonus could be explained by the fact that the "tonizing" on the centers of gaze in this case was chiefly restricted as far as the nonoptical stimuli were concerned; a condition remaining as it was before and soon after birth.

2. Since adjusting movements may be regarded as developing chiefly by inhibition of optical stimuli which do not attract the attention, one may assume a disturbance in the development of the pathways along which stimuli from other parts of the retina are inhibited. Perhaps this assumption finds support in Cogan's report that a normal child usually blinks his eyelids before making a voluntary eye movement, as if he would thus aid this inhibition.

Burg. v. Royensingel 1.

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Ophthalmic Miniature

Ann Whalley, aged 23, came under my care nine years ago, when 14 years old, having congenital cataracts of both eyes, on which I operated with success. Some circumstance induced her mother to go out of town suddenly, before the eyes were quite clear, and I did not see her again until the 11th of March last, when a small portion of capsule appeared to impede vision at the lower part of the pupil of the right eye, the left being free. Supposing that the removal of this portion of the capsule would improve her sight I proposed it to her, and on doing it, I found, to my great surprise, that the lens had been reproduced, and was quite transparent. It became, of course, opaque, and now is dissolving in the usual manner.

G. J. Guthrie:
The operation for the extraction of a cataract from the human eye, etc., 1834, p. 14.

NOTES, CASES, INSTRUMENTS

SIMPLE REPAIR OF EYELID MARGIN DEFECTS*

D. RALPH MILLARD, JR., M.D.
Miami, Florida

The eyelid, an exquisitely delicate structure, endowed with a comb of cilia, lubricated lining, intrinsic and extrinsic musculature, and thin elastic skin, has been intricately engineered to protect the staring eyeball. It is little wonder that, when faced with a benign or malignant lesion involving a good portion of the eyelid margin, the surgeon is apprehensive. Even when the safest approach is a clean excision, fear of mutilation of this vital structure has caused the surgeon to seek escape in procrastination or in referral to a roentgenologist. A simple answer could be in a plastic trick which would allow adequate excision and yet avoid any visible deformity.

JUGGLING THE SCARS

For lesions of the eyelid margin which require full-thickness excision of one third or less of the eyelid, the usual attack has been the "V" or pie-wedge excision which invariably ends up with a vertical scar and eventually a notch or ectropion. This led O'Connor to the block excision which produced the same vertical scar but reduced the incidence of notching or ectropion.

A vertical scar of the eyelid, with or without contracture, is distasteful and should be avoided. This is possible by following the principle of not putting all eggs in one basket or rather not putting all scars in one direction. The mucomuscular layer can be closed in the usual vertical (side-to-side) manner but the skin closure must be horizontal. Straith described one case in which he obtained a horizontal closure of the eyelid skin by an extension of his excision beyond the confines of the eyelid and curving down into the cheek. Although in principle correct, the method seems slightly more radical than necessary. The ideal would be an eyelid repair which was simpler with a scar less visible (fig. 1).

Fig. 1 (Millard). Result two and one-half years following excision of basal-cell carcinoma of the middle margin of the lower eyelid. The defect and the method of closure are shown in Figure 2.

VERTICAL MUCOMUSCULAR CLOSURE

After the lesion and an adequate margin (up to one third of the lid) has been excised (fig. 2-A) the conjunctiva and muscular layers are approximated in a vertical direction. A 4-0 silk suture brings the defect together at the ciliary margin and facilitates the mucomuscular closure (fig. 2-B). Subconjunctival sutures of 5-0 chromic catgut which also pick up good bites of orbicularis muscle will achieve a neat mucomuscular approximation without exposing sutures on the conjunctival side. If as much as one half of the eyelid margin must be sacrificed, a lateral canthoplasty somewhat in the manner used by Reese for his greater resection will be necessary to advance the ciliary margin, muscle, and conjunctiva to enable this vertical closure.

* From the Department of Surgery, University of Miami School of Medicine, and Jackson Memorial Hospital.

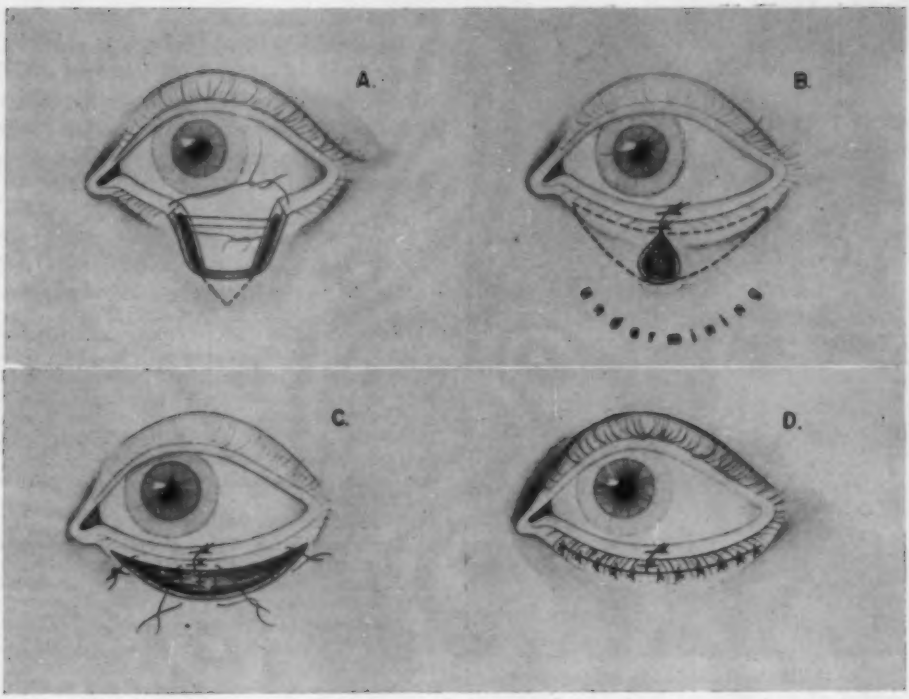

Fig. 2 (Millard). (A) Defect following excision. The dotted triangle indicates a possible extra excision of muscle and conjunctiva to assist in the vertical mucomuscular closure. (B) The marginal and vertical mucomuscular closure has been accomplished. The dotted line indicates the excess skin to be excised after the eyelid skin has been freely undermined. (C) Simple closure. (D) Hidden scars, without deformity.

Horizontal skin closure

A horizontal incision in the eyelid skin just under the lower lid lashes or just above the upper ones is then made and extended out on either side often as far as each canthus. The eyelid skin is then freed from the underlying muscle by generous undermining and the center point of the skin defect is lifted and stitched to the ciliary margin. The excess eyelid skin which is now overlapping on either side can be excised as a triangle along the lower dotted line (fig. 2-B, C). This produces a relatively straight advancing skin edge which can be raised (lower lid) or lowered (upper lid) like a curtain and hung with sutures along the ciliated margin in a horizontal and hidden position (fig. 2-D). The moderate side-to-side tightness of the vertical mucomuscular closure creates a relatively tight span which can withstand the drag of the lifted lid skin. Thus a tendency toward ectropion is counteracted. The Wheeler halving procedure is not deemed necessary here inasmuch as the through-and-through vertical scar has been avoided.

Evaluation

This method has been found effective in both upper and lower lids in margin excisions sacrificing as much as one third. If one half of the lid requires excision then a canthoplasty will be necessary to allow the vertical mucomuscular closure. No ectropion, depression, or notching has been noted and, after a few weeks, it is difficult

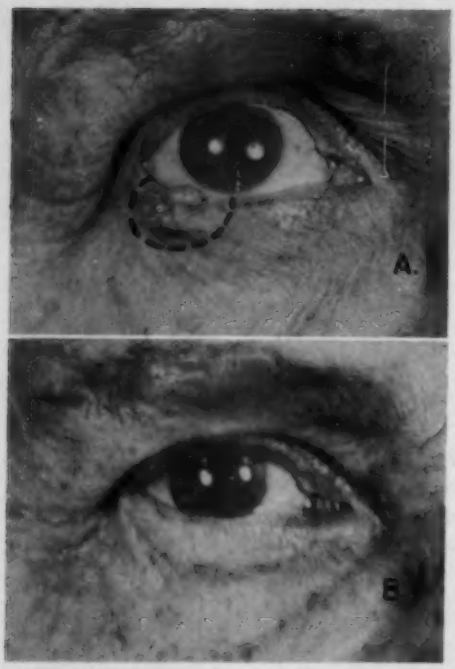

Fig. 3 (Millard). (A) Basal-cell carcinoma marked off in preparation for wide excision. (B) Two years after closure as described.

to find evidence of the surgery (fig. 3). Although this technique is applicable to patients of all ages, it has its greatest usefulness in the aged in whom malignant eyelid lesions are more common and in whom there is laxity and skin in abundance, wrinkles, "bags," and all. In fact, the repaired eyelid may so outshine its sad and sagging fellow that a minor matching plastic procedure may be required to prevent the two from looking like father and son.

Discussion

Dr. Edward E. Moore
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The traditional halving procedure for removal of lid-margin tumors is an excellent method but there are several objectionable features encountered in its use. An additional amount of lid margin tissue is sacrificed in order to produce a satisfactory closure. It is necessary to do an external canthotomy in order to make up for the lost tissue. "Splitting" the lid margin sometimes causes trichiasis even in the most careful hands.

Dr. Millard has devised a technique which is free of these objections. With his procedure, a minimum of lid margin is lost and external canthotomy is unnecessary unless more than one third of the lid is excised.

This method has been used by me in six cases with excellent cosmetic and functional results. The procedure is much simpler than the lesser resection. Trichiasis has not been encountered nor has there been any notching of the lid. The removal of a generous amount of skin from the lid does not produce an ectropion. This new method is recommended as a definite improvement over the presently accepted technique for excision of tumors involving the lid margin.

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SAFEGUARDS IN CATARACT SURGERY

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In a future Utopia all cataract extractions will be performed by senior surgeons in special hospitals with highly trained assistants. The present laudable efforts to restrain the "occasional surgeon" are the first steps toward this ideal but, when the latter has been suitably controlled, there will still remain the problem of providing perfect working conditions for the experienced operator. The occasional surgeon working in an eye hospital is often less of a menace than the expert called upon to work with untrained help.

There is no doubt that such a situation frequently arises. Small communities distant

from large centers usually have excellent general hospitals whose operating room staffs cannot realize the importance of experienced assistance in eye surgery. Similar conditions are encountered in sanatoria, state institutions, in foreign missions, and in warfare.

During the course of many years of surgery in a state hospital for the insane, I have developed an unusual degree of self reliance while working with patients whose conduct is often unpredictable. The special precautions which have been found necessary and the methods of briefing assistants with no previous knowledge of eye surgery may be of interest to others who find themselves in similar circumstances.

The essential points in this procedure, that is, the use of colored beads to identify the sutures and to close the wound, and the retention of a sclerocorneal bridge may be only modifications of methods described by others. However, a review of the literature has disclosed no sources to which credit could be given.

As the removal of sutures in patients of this type presents a special problem, the use of absorbable material is essential. However, catgut is notoriously difficult to see and control, particularly when contrasted with a background of presenting vitreous. To overcome these disadvantages it has been found useful to identify each suture by fixing a small colored bead at each of its ends. This serves three purposes. In the first place, the beads keep the ends of the sutures flat and readily identifiable. Secondly, each suture can at all times, even in the tangle which always develops in an emergency, be recognized by the color of its bead. Thirdly, the beads act in the place of the conventional knot for emergency wound closure.

The beads have several advantages over knots. A single knot on a catgut suture can easily be embedded in the cornea or even pull through. There is less tendency for the sutures to break, as the degree of traction on the suture can be controlled by watching the lines of tension on the cornea which radiate from the bead, much as a helmsman watches the luff of a sail for "flutter." This is of considerable importance with an unskilled assistant who can be instructed to watch for these tension lines and relax his hold as they appear.

The beads measure only two mm. in diameter and have a lumen which just admits the needle of a No. 790 Ethicon chromic catgut suture. They are obtainable at any hobby shop as components of an Indian beadcraft outfit or direct from the Walco Bead Company, 37 West 37th Street, New York, New York.

Three chromic sutures are prepared before use by a nurse who attaches a red, a white, and a blue bead, respectively, to one end of each, using a single hitch.

A fornix-based flap is first dissected up. A Graefe section is then made and, after the counter-puncture, the knife is carried upward but withdrawn before the section is completed, leaving a small corneoscleral bridge. The two openings thus left in the cornea are enlarged if necessary with scissors. At the 10- and 2-o'clock positions, a peripheral iridotomy or iridectomy is performed.

The suture with the white bead is now passed through the corneal bevel on the nasal side and out through the sclera. As the needle passes through the scleral margin the assistant holds a similar white bead in

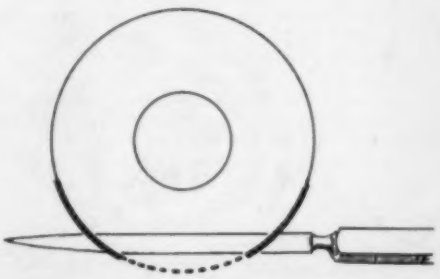

Fig. 1 (McGrath). Knife withdrawn before completion of Graefe section (dotted line), leaving corneoscleral bridge.

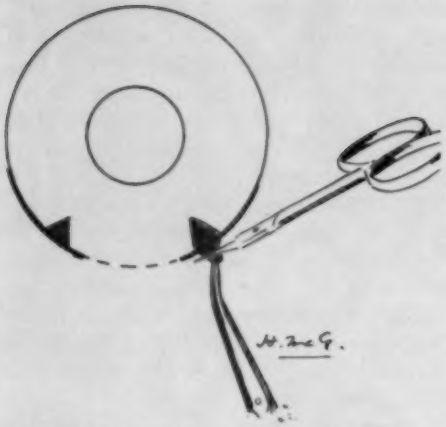

Fig. 2 (McGrath). Peripheral iridotomy or iridectomy at the 10-o'clock and 12-o'clock positions through pockets thus formed.

a Verhoeff capsule forceps and the needle is passed through it. The bead is tied to the suture. There is now a like colored bead at each end of the suture. A similar procedure is carried out through the temporal wound and a blue bead threaded to the suture. Blue always indicates the temporal side and white the nasal, so that either side of the wound can be closed in case of emergency.

At this point, before the wound is opened finally and completely, it is possible to pause for deliberation and to explain to the neophyte assistant who may be assisting at his or her first eye operation just how the sutures are to be handled in case of emergency.

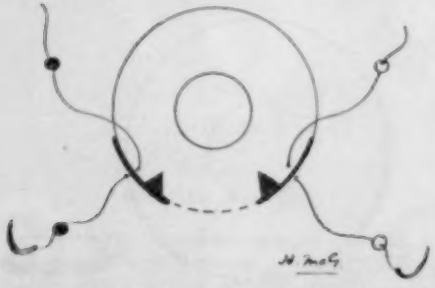

Fig. 3 (McGrath). Sutures with colored beads attached—blue on nasal side, white on temporal—introduced through nasal and temporal sections.

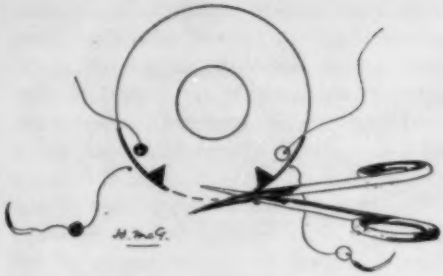

Fig. 4 (McGrath). Wounds closed against beads and corneoscleral bridge severed with scissors.

He is shown how to pull on the white bead or the blue, or both, to obtain temporary closure.

He is also instructed to close the wound against the lateral beads as the lens equator passes through the wound. When this is clearly understood, the corneoscleral bridge is severed with scissors and he closes the wound against the lower beads. The suture at the 12-o'clock position with the red bead is now inserted and threaded through another red bead. The lens is then delivered. It is clearly understood by the assistant that the red beaded suture is to be handled only by the surgeon, who also takes care of the speculum. The assistant is responsible for the blue and the white lateral sutures. Under these conditions bulging or even presentation of the vitreous can be regarded with composure.

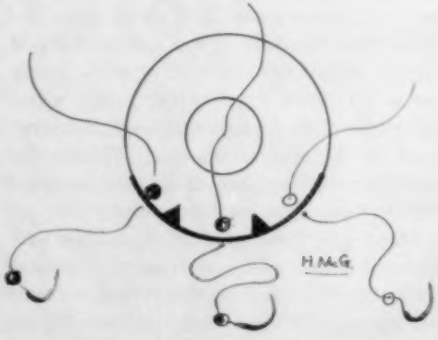

Fig. 5 (McGrath). Suture threaded with red bead inserted at the 12-o'clock position.

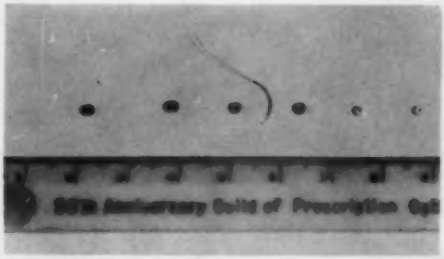

Fig. 6 (McGrath). Comparative sizes of needle and beads.

After the lens is delivered, the toilet of the wound completed, and any complications dealt with, the beads at the corneal side are picked up individually, the sutures tied, and the beads cut off.

Only those who have been unfortunate enough to have to try to identify the appropriate ends of 6-0 catgut sutures in an effort to tie them while they float on an expanding balloon of vitreous will appreciate the comfort the colored beads provide in such trying conditions. To those ophthalmologists who on occasion find themselves in lonely isolation in the face of disaster with willing but inexperienced assistance, some or all of these details may be of value.

Summary

A method of cataract extraction adapted especially for the surgeon operating alone without skilled assistance on an unpredictable patient is described. It consists of the use of different colored beads threaded onto catgut sutures. The beads provide ready identification for the untrained assistant, and also serve as a means of temporary closure of the wound. A further safety factor is provided by the retention of a corneoscleral bridge after the Graefe section.

41 Maple Avenue.

PETECHIAL HEMORRHAGES*

Following treatment of scleritis with cortisone

Charles A. Turtz, M.D.
and
Arnold I. Turtz, M.D.
New York

Although steroids have proved valuable additions to ophthalmic therapeutics, their increased and widespread application have produced unusual side reactions.

Case report

Mrs. L. W., aged 48 years, a school teacher, consulted us on November 12, 1956, and gave a history of a red painful right eye, without any secretion, for three days, following a head cold. She recalled that she had had a red painful eye several weeks previously, which had cleared up after warm saline applications. Her past medical history was irrelevant except for recurrent bursitis of the right shoulder which was relieved by salicylates and local heat.

Examination revealed a healthy appearing woman who had visual acuity of: R.E., 20/50; L.E., 20/40, correctible to 20/25 in each eye with hyperopic astigmatic lenses. There was folliculosis in both lower fornices, more marked in the right eye. In that eye there was a deep lilac-colored, localized area of scleral injection, temporally, which did not blanch after adrenaline instillation. The pupils reacted actively to light and accommodation. There was no muscle unbalance.

Slitlamp examination revealed an old posterior synechia in the right eye at the 4-o'clock position. The cornea and lens were clear. Funduscopic examination revealed nothing remarkable except for slight changes in the smaller arterioles. Fields and tension were well within normal limits.

A diagnosis of scleritis was made. Two-percent solution atropine sulfate twice daily was prescribed together with 2.5-percent cortisone solution every two hours. Sodium salicylate and local warm saline applications were also advised. Re-examination three days later revealed that the injection of the eyeball had cleared up, the eye was white, and the patient claimed she was free from pain. The synechia was torn loose and the pupil was widely dilated. There were, however, hundreds of pinpoint petechias present on the bulbar and tarsal conjunctiva of the right eye. Cortisone was then discontinued. When she was seen three days later there was a recurrence of the scleritis and the petechias were absent. The use of cortisone was resumed;

* From the Department of Ophthalmology, New York Medical College.

there followed a return of the petechias. Medication was gradually withdrawn and the scleritis disappeared. Cortisone was discontinued and there was again complete absorption of the petechias.

Summary

Local use of cortisone presumably produced petechias of the bulbar and tarsal conjunctiva. These were completely absorbed after discontinuing the use of this drug. We could find no plausible explanation for this unusual side reaction.

525 Park Avenue.

OSTEOMA OF THE ORBIT

B. M. Chatterjee, F.R.C.S.
Calcutta, India

Osteoma of the orbit is rather an infrequent tumor. Of 109 cases of tumors arising primarily in the orbit, Reese,[7] in 1941, found osteoma in only one case. Forrest[8] reviewed 184 primary orbital tumors and found osteoma in only five cases. Benedict[1] reported that 38 of 740 tumors or tumor-like lesions of the orbit at the Mayo Clinic were osteomas.

Osteomas are benign tumors which, according to Duke-Elder,[2] arise from paranasal sinuses as a result of disturbances or displacements of cartilage in the process of development, the stimulus for new growth being chronic infection or unsuspected fracture. Fetissof,[4] in 1929, suggested a periosteal origin of osteoma. Whatever may be the mode of origin, the most common site is the frontal sinus and then the ethmoid cells, the maxillary antrum, and the sphenoid.

Parsons[6] (1905) divided these tumors histologically into two main types: hard, eburnated, or ivory osteoma (70 percent) and soft or cancellous (12 percent). The rate of growth of these tumors is very slow, so pain and diplopia are not outstanding features. The following is the report of a typical case of an ivory osteoma arising from the ethmoid.

Case report

P. L. N., a man, aged 34 years, attended the S.S.K.M. Hospital, Calcutta, on December 7, 1956, with the complaint that his left eyeball, which had been slightly pushed forward for the last 13 years, recently became more prominent. He also complained of occasional vague pain in the left eye but there was never any visual disturbance either in the form of double vision or diminution of sight. There was a past history of frequent attacks of cough and cold for the last 13 years but there was no definite history of any trauma to the left orbit.

On examination, the left eye was found to be displaced slightly downward and outward, with a proptosis of three mm., measured by the Reyner exophthalmometer. The ocular movements were normal except for a slight diminution of movement of the eyeball upward and inward. There was no congestion of the conjunctiva or edema of the lids. The cornea and media were clear. Pupil reactions were normal and there was no abnormality in the fundus. Vision was 6/9 and no field defect could be detected. On deep palpation along the orbital margin, a suggestion of a hard mass could be obtained deeply situated at the upper inner angle. Intraocular pressure was normal.

The skiagram of the left orbit, anteroposterior view (fig. 1), showed the presence of a dense white area in the ethmoidal region at the upper and inner angle. From this area, a dense white globular mass on a narrow stalk projected into the orbit.

On lateral view (fig. 2), it was evident that there was no extension of the dense white mass either inside the frontal sinus or intracranially. Radiologically, the mass was diagnosed as an ivory osteoma arising from the ethmoid. A skiagram taken on June 25, 1948, about nine years ago, showed practically the same picture (figs. 3 and 4).

The right eye was normal, with vision of 6/6. All other laboratory investigations including Wassermann test were negative.

Operation. Since there was mild degree of proptosis and the patient was complaining of vague pains, surgical removal of the tumor was decided. Although there was apparently no intracranial extension of the tumor, a neurosurgeon* assisted me in removing the tumor. On December 12, 1956, under general anesthesia, an incision was made for about 1.5 inches along the upper and inner margin of the left orbit. The skin, subcutaneous tissue, and orbicularis oculi were incised and periosteum was exposed a little beyond the orbital margin. By incising the periosteum, an attempt was

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* I wish to thank Dr. A. K. Bagchi for assisting me in this case.

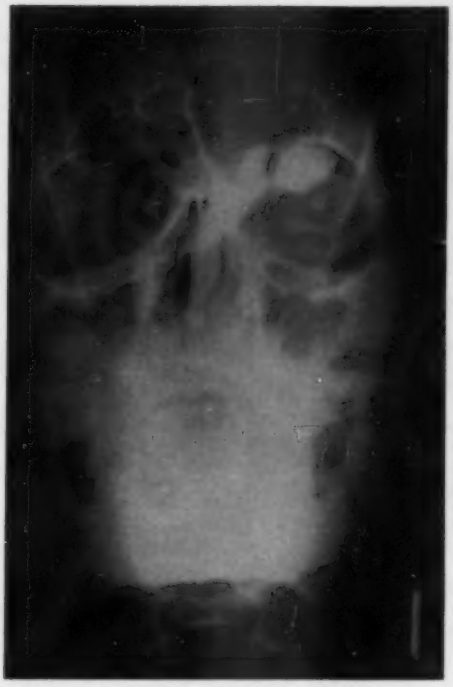

Fig. 1 (Chatterjee). Anteroposterior skiagram of left orbit.

made to reach the tumor subperiosteally. However, since the periosteum was found to be inseparably adherent to the tumor, this method of getting access to the tumor had to be abandoned. As an alternative, the orbital septum was incised along the orbital margin and the orbital contents were exposed.

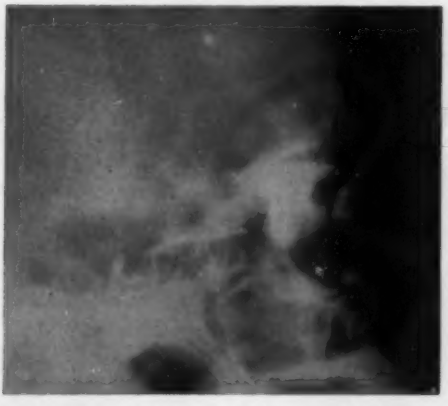

Fig. 2 (Chatterjee). Lateral skiagram of left orbit.

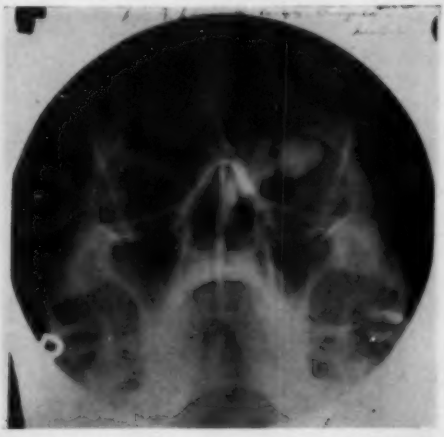

Fig. 3 (Chatterjee). Anteroposterior skiagram of left orbit taken nine years before present examination.

On entering the orbit, the reflected tendon of the superior oblique was also divided between two preplaced sutures. When the orbital fat was retracted, a hard white mass with a knobby surface, about 0.75 by 0.5 inch in diameter, was seen to project inside the muscle cone extending from the ethmoidal region and passing outward underneath the superior rectus. The mass had a broad base with a narrow

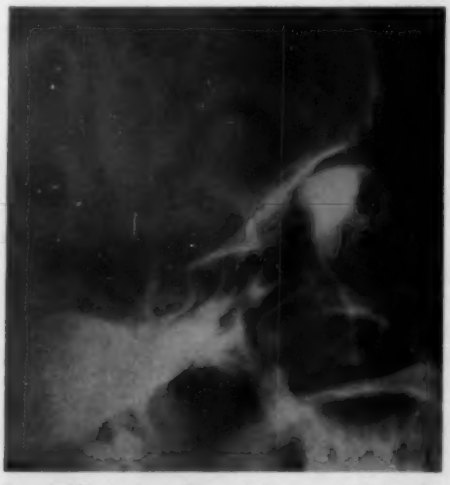

Fig. 4 (Chatterjee). Lateral skiagram of left orbit taken nine years before present examination.

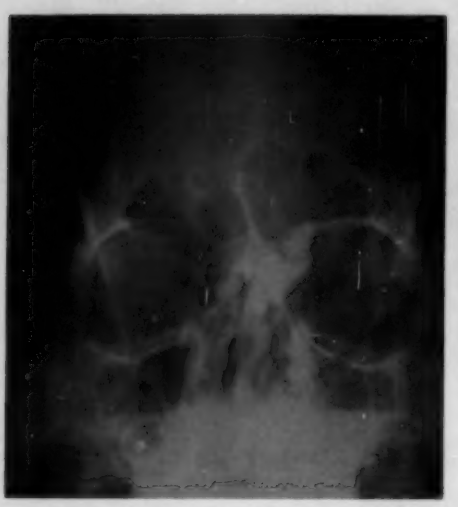

Fig. 5 (Chatterjee). Anteroposterior skiagram of left orbit after removal of tumor.

neck and a rounded head which was lying behind the eyeball but not pressing the optic nerve.

The tumor proved to be extremely hard and the narrow stalk could only be nibbled off with extreme difficulty. The globular head came out easily. Since any attempt to remove the base of the tumor would have produced wide destruction of the orbital wall, leading to almost complete removal of the lateral ethmoid, this portion was left. The tendon of the superior oblique was sutured and the wound was closed in layers. The recovery was uneventful. Although a slight displacement of the eyeball persisted, proptosis disappeared completely.

A skiagram of the left orbit taken after the operation is shown in Figure 5.

Pathologic examination. Histologic section of the tumor showed the typical picture of an ivory osteoma composed of concentric layers of compact bone devoid of blood vessels (figs. 6 and 7). It is said that an orbital osteoma is usually covered by the mucous membrane of the paranasal sinus from which the tumor originates. But, microscopic examination did not reveal any trace of such mucous membrane; probably it was atrophied.

Fig. 6 (Chatterjee). Section of tumor (low power).

Fig. 7 (Chatterjee). Section of tumor (high power).

Discussion

Consideration of this case report makes it evident that the osteoma, proved histologically, originated at an unknown date from the ethmoidal sinus, without any obvious exciting cause. Symptoms started to appear about 13 years ago. Its presence was detected radiologically nine years ago, after which time it remained stationary.

The minimal eye signs in this case were obviously due to the extremely slow growth of the tumor which accommodated itself to the orbital contents without disturbing the function of any important structure. To treat such an orbital tumor, it was imperative to decide whether operative removal was indicated and, if so, which type of approach could be used to the best advantage. Vague pains and slight proptosis, although minimal symptoms, were considered to be sufficient indications for operative interference.

According to Benedict,[1] as the osteoma lies underneath the periorbita, it is convenient to approach the tumor by incision through the skin and periosteum anterior

to the surface of the tumor. Rizzuti[8] and Damato[2] also adopted the subperiosteal approach. In the present case this method failed, so approach was made through the second surgical space between the periorbita and the muscle cone.

Considering the imperceptible growth of the tumor, it seems improbable that it will recur; however, the patient is still under observation.

244, Lower Circular Road (20).

ACKNOWLEDGMENT

I am indebted to Lieut. Col. N. C. Chatterjee, surgeon superintendent of the S.S.K.M. Hospital, for permitting publication of this paper.

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CONGENITAL ANTERIOR STAPHYLOMA WITH HYDROCEPHALUS*

A. D. GROVER, M.S.
Aligarh, India

CASE REPORT

Meena, aged 28 days, a girl, was brought on February 5, 1953, with the complaint of the absence of the black portion of the eyeballs and the inability to see since birth.

Family history. The parents had been married for three years. The patient was the first child; full-term, normal delivery; weight, six lb. The mother had a high fever for three days during the fourth month of pregnancy, but no exanthema. There was no history of any venereal disease.

Personal history. At the time of birth, the central black part of the eyeballs was not seen and in its place there was a protruding mass. The area round about the bulging portion of the eyeballs became bluish on the third day after birth and has been so since then.

Examination. There was a three to four-mm. protrusion of an anterior staphyloma through the palpebral fissure of the right eye. The size of the eyeball was normal. The surface of the staphyloma was lobulated especially in its upper part. The sclera for four to five mm. around the limbus was bluish.

In the left eye, there was a two to three-mm. bulging of an anterior staphyloma in the upper two thirds, while in the lower one third the cornea was

Fig. 1 (Grover). Appearance of patient.

* From the Gandhi Eye Hospital, Muslim University.

opaque and flat. The sclera for four to five mm. around the limbus was bluish. The size of the eyeball was normal (fig. 1).

Investigations. Conjunctival smear, negative; Mother's Kahn and Wassermann reactions, negative. Examination of the mother showed subacute cervicitis with erosion of the cervix. The vaginal smear showed a few pus cells but no organisms.

Follow-up. The condition of the patient's eyes remained almost the same but the general health of the child deteriorated. Ten days after the first examination, the fontanelles gradually became large, tense, and bulging and the child started showing signs and symptoms of hydrocephalus. The circumference of the head gradually increased to 27 inches. The child died at the age of 11 months.

Discussion

Congenital anterior staphyloma is a rare condition. Etiologically, it probably exists in two forms: (1) truly developmental aberration, and (2) the result of an intrauterine inflammation. The theory of intrauterine inflammation was originally advanced by Sonnenmayer (1840) and, in many cases, the condition bears a striking analogy to an ulcerative process which has advanced to perforation. A case studied in detail by Parsons (1904) showed definite signs of infection, such as occurs in postnatal life. Clausen (1922) has described a case of congenital staphyloma with signs of inflammation. In some recorded cases the condition was bilateral. A few cases showed an anterior staphyloma in one eye and phthisis bulbi in the other; this points to a transamniotic infection and may mean that the phthisical eye has shrunk as a result of more severe panophthalmitis than that of its fellow which merely became staphylomatous. In some cases the infection may be endogenous, that is, transplacental, and the resulting staphyloma may be a further development of an "internal ulcer."

On the whole the evidence points to an inflammatory origin in most cases of congenital staphylomas, whether transamniotic or transplacental is uncertain.

On the other hand, the cases in which inflammatory changes are absent and some other developmental anomalies, such as adherence of lens or iris or pupillary membrane to the back of the cornea, are present, can be explained by the theory of "maldevelopment." In Peter's view the aberration primarily concerns the formation of the lens vesicle from the surface epithelium; while in Collins' view, a nondifferentiation of the mesoderm intrudes itself between the lens vesicle and the surface ectoderm.

The patient herein presented had anterior staphyloma in both eyes since birth. She developed hydrocephalus at the age of one month. In some cases, chronic hydrocephalus in children is due to excessive secretion by the choroid plexus or ventricular ependyma as a result of chronic inflammation (Romanis and Mitchiner). It is quite probable that in this case the anterior staphyloma and chronic hydrocephalus were the result of an intrauterine endogenous (transplacental) inflammation.

Gandhi Eye Hospital.

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SOCIETY PROCEEDINGS

Edited by Donald J. Lyle, M.D.

CHICAGO OPHTHALMOLOGIC SOCIETY

November 18, 1957

Dr. Frank Newell, *President*

Successful Hummelsheim operation

Dr. Don Smart and Dr. Daniel Snydacker presented the case of a 36-year-old man with a complete right lateral palsy of unknown origin. In March, 1956, a resection of the right lateral rectus and a recession of the right medial rectus were performed. The original 50 prism-diopter esotropia was reduced to 25 prism diopters by this procedure and in May, 1956, a five-mm. recession of the left medial rectus was performed without subjective or objective improvement. Therefore, in November, 1956, a Hummelsheim operation was performed on the right eye. Since this time the eyes have remained straight for distance and near and the right eye can be abducted 20 to 30 degrees.

Provocative tests in glaucoma

Dr. Frederick Blodi (Iowa City, Iowa): Any test that will help us to make an early diagnosis of glaucoma is worth further study. Provocative tests if used judiciously and applied intelligently will be of such help.

The reliability of a certain test has to be known. This is the expected percentage of positive results in a series of patients with untreated early glaucoma. This figure should be high or the test is impractical. The analytic evaluation of a test will reveal the mechanism by which the test produces an increase in intraocular pressure. An understanding of this mechanism will allow us to choose a specific test for a specific type of glaucoma.

The positivity of a test is usually judged by the increment of intraocular pressure. After the water-drinking test an increase of more than 6.0 mm. Hg is highly suspicious for glaucoma. After the Priscoline test an increase of 9.0 mm. Hg is still within physiologic limits. The final value reached after a provocative test is not of paramount importance as it depends too much on the original intraocular pressure.

Tonographic studies before and after the water-drinking and the Priscoline tests seem to indicate that the increase in intraocular pressure is due to an increased inflow of fluid into the eye. This increased flow can be partially inhibited by the administration of Diamox one hour before the test.

The provocative tests emphasize the importance of certain hygenic measures in glaucomatous patients. They should not wear a tight collar nor work with their head bent down, as in gardening. They should avoid drinking large amounts of liquids and they should cut down on coffee, not because of the caffeine but because of the amount of fluid.

The water-drinking test and, if that is negative, the Priscoline test should be used in chronic simple glaucoma. In closed-angle glaucoma the darkroom and the mydriasis tests are of greater value.

Mechanics of corneoscleral wound closure

Dr. William H. Johnston presented a study of closure of the incision made for cataract extraction. He compared the radial suture (McLean) with the mattress suture (Alvis and Bick). He showed that both sutures, if improperly placed, cause a posterior gaping of the wound and that although a mattress suture is superior in this respect it is more difficult to remove and doubles the number of loops of material at the time of operation. The most important factor in preventing posterior gaping of the corneal

wound is a shelving incision. With this type of incision the pressure of the intraocular contents coapts the wound edges ever tighter. Dr. Johnston believes that the ideal closure is a well-shelved incision closed with several radial appositional sutures.

Discussion. Dr. Kenneth L. Roper: Dr. Johnston has recently had two serious postoperative hemorrhages in cataract patients. Hemorrhage is but one of several complications that can occur as a result of defective wound healing. It is too often attributed to bad behavior on the part of the patient. Dr. Johnston did not place his two cases in this category but instead has become interested in the causes of defective wound healing. He will benefit as well as his patients because of this aroused curiosity.

In my own experience, hemorrhage into the anterior chamber is rarely traumatic in origin but usually develops spontaneously in cases in which a suture has been placed too deeply on the scleral side of the incision. Vail has written extensively on hyphema after cataract extraction and his conclusions are well known to all of you. The lower incidence of hyphema that I have might well be ascribed to the use of multiple corneoscleral sutures which he advocates as an important measure against hyphema.

Faulty closure of the cataract wound occurs when the lips of the incision are not in proper apposition. This can take place either with or without tissue being included between the edges of the incision.

Many investigators, more recently Dunnington, have shown how basic principles of wound healing quite thoroughly establish the value of a limbal incision with a conjunctival flap. The highly vascular nature of episcleral tissue causes prompt closure of the anterior part of the wound, while the conjunctival covering tends to prevent epithelium from growing between the edges of the incision.

Such studies also point out that approximation of the deeper layers of the wound is essential.

Superficial sutures tied too tightly tend to cause gaping of the posterior surfaces. This can lead to spontaneous loss of the anterior chamber on or about the fifth day, with or without incarceration of the iris or vitreous. This predisposes to later complications such as persistent striate keratopathy, cystoid cicatrix, and vitreous traction on the retina which further leads in some cases to macular edema or possibly retinal detachment.

If sutures are placed too deeply, gaping of the anterior layers occurs. If a conjunctival flap has been employed, the vascular wedge of episcleral tissue is large and any separation of the posterior layers will be followed by bleeding into the anterior chamber.

Overriding of the edges of the wound will occur when the suture is too superficial on the opposite side.

In the case of a mattress-type suture, unless it is most accurately tied, an inversion of the scleral part of the incision occurs. A similar result will occur with the use of a single appositional suture if the scleral bite is too far away from the edge of the incision.

In my hands faulty wound healing from these causes has been largely overcome by the use of three nonperforating appositional sutures inserted to a depth preferably less than one-half the thickness of the tissues at about the junction level of the outer and middle third. It can be readily seen that, with sutures so placed in wounds more or less oblique and valvular and combined with a conjunctival flap, cohesion sufficient to withstand normal intraocular pressure results, and the anterior chamber is restored within a few minutes.

Dr. Johnston has obviously been giving a lot of thought to improving his own cataract technique and I asked him to let me know how he has done his recent cases. He was kind enough to write me a few days ago and to include a copy of his paper. He also sent along a list of questions which he hoped I would answer. I shall now attempt to do so.

Dr. Johnston prefers a preplaced shallow groove; four preplaced McLean sutures; and a very bevelled keratome incision.

First of all let's give credit where credit is due. For the benefit of the younger men and some of the older men as well, Williams, in 1865, was the first to employ a suture to close the cataract incision. His suture was placed in the cornea after the extraction of the lens.

Mendoza, in 1888, was the first to make a preliminary groove in the cornea and to insert the ends of a double-armed suture in either lip prior to the incision.

All that McLean did in 1940 was to move the groove back toward the sclera so that he could incorporate the conjunctiva.

The importance of a conjunctival flap is so generally conceded that comment is unnecessary. Dr. Johnston states that the limbus-based flap bothers him. It obstructs his view of the chamber and is a hindrance in making his keratome-scissors section. This is one of my objections to it. I also feel that this type of flap is handled too much during the operation, resulting in tears and also requiring the insertion of many interrupted sutures. Dr. Johnston wrote that he might adopt a fornix-based flap and I advise him to do so.

Dr. Johnston states that he prefers 6-0 black braided silk on Greishaber needles. In the first place, it seems to be generally agreed that suture materials themselves—silk or catgut—as now available for cataract surgery are satisfactory. The second factor, which is of extreme importance, is the quality of the needles available to us today. There is no longer the need to depend on the Greishaber needle which meets all the eye surgeon's requirements in terms of sharpness, polish, and performance, with one important exception. The fact that it is still an eyed needle necessitates that two strands of suture material be pulled through the tissue. Less tissue damage and a smaller hole occurs with the single strand attached to a swagged needle.

Dr. Johnston inquires as to whether it is easier to make a bevelled section with a keratome than the Graefe knife. It is well known that the usual keratome incision is bevelled, so that re-established intraocular pressure tends to close the wound more tightly.

Dr. Johnston also asked how does one decrease the bevel as the 180-degree meridian is approached. I know of no reason why one should aim to do so. If, on the other hand, the bevel is continued when enlarging the wound, a firmer closure is procured, so that the wound is less likely to leak.

I think it is important to point out that if a long bevel is made, as advocated by Atkinson, it is essential to tumble the lens. In the event of capsule rupture it is more difficult to express lens matter. I prefer an incision with a shorter bevel than advocated by Atkinson and placed well back at the limbus, which permits safe delivery of any type of lens with which one is confronted.

Dr. Johnston asks "how does one make certain, when a fornix-based flap is used, that tags of conjunctiva do not get into the wound." My answer to this question is—first of all—careful, meticulous dissection of the flap with a loupe, making certain that there are no conjunctival tags left hanging to the cornea. Also, as I have already mentioned, I place my incision well back of the conjunctiva-cornea junction line.

Dr. Johnston also inquires as to whether or not right and left scissors for enlarging the corneal section is superior. It definitely is.

I personally use the keratome and scissors and feel that this major objection is best answered by the use of proper scissors for enlarging the section. I have had made for me special right-sided and left-sided angular scissors which has proved extremely helpful for enlarging the corneal section. The scissors is used as a pair. The blades are angulated, 14 and 16 mm. long, with smoothly rounded points like a spatula, enabling easy introduction into the anterior chamber without in-

jury to the corneal endothelium or iris. The blade which is introduced into the anterior chamber on both the right-sided and left-sided scissors is the lower and longer blade, permitting a continuation of the section at the same level or plane on both sides of the keratome incision and without having to reinsert the scissors into the chamber with each snip.

And now back to Dr. Johnston's original problem—what caused the severe hemorrhages in his two cases. At least he did not attribute either to bad behavior on the part of the patient. Instead, he has become interested in the causes of defective wound healing. He is to be commended for having this aroused curiosity. Through it he has learned much and his patients are going to be benefited by it. And the deeper he gets into the problem of suturing in cataract surgery, the more convinced, I am sure, he will become that there is nothing new about it under the stars.

Dr. Derrick Vail: Mr. President, fellow members, and guests. Dr. Johnston was very kind to me, but I'm not so sure about Ken's remarks. At any rate, I don't know whether we need a defense or not. I want to congratulate Dr. Johnston for a very careful study of the factors that enter into the posterior gaping of the wound, which, I agree with him, is a big factor in the incidence of hyphema. There is an awful lot to say about this. In the first place, the incidence of hyphemas is one of the very thorny problems to determine; in the literature it varies all the way from 0.10 percent up to almost 39 percent, and you have to strike an average. The average comes up to 12 percent now, I suppose. When I made the original study of over 3,000 cataract cases—patients who had cataract operations—including about 1,200 in the literature, the over-all figure that I had was six percent. In a recent study of controlled cases where I wanted to see whether Diamox had any effect on the healing, the incidence was quite high, about 23 percent. I don't quite know in my own mind the answer to that. The technique was considered to be about the same. I think we may have used cortisone a little sooner in those cases because cortisone, in the original series, was not available.

My partner, Dr. Shoch, has had an excellent run of cataract patients without much in the way of hyphema to bother him. I do know that the proper sutures he uses have cut down the incidence and there are papers in the literature to emphasize that point. A colleague in Wisconsin, Fred Davis, tells me that he has done 200 consecutive cataract cases without a single hyphema, using three or four catgut sutures. I listen to Fred Davis with a great deal of respect because he has had, of course, enormous experience. That is just one proof, you might say, that proper suturing does have an effect on hyphema and I think Dr. Johnston has shown us why.

There are any number of unsolved problems in this fascinating subject. In the first place, the hyphema itself can vary from a drop or so of blood in the anterior chamber, which is trivial, to the desperately bad cases where blood fills the anterior chamber and, also, the vitreous. In a number of cases, eyes have been lost as a result of these serious hemorrhages. I think probably Dr. Johnston's two cases may fall into this category. I have a feeling that those cases in which we have massive intraocular hemorrhages, which we consider as an extensive hyphema, actually are a combination of the ordinary mechanism of hyphema which is wound rupture, plus a subchoroidal hemorrhage that was not sufficiently powerful to break through the sutures and extrude all of the contents. The proof of that statement would probably lie in some work that Bernard Samuels did a number of years ago in the study of eyes enucleated for hopeless blindness and secondary glaucoma at the Eye and Ear Infirmary in New York. He showed subchoroidal hematomas in a number of these eyes.

So I think we have to go back to the first principle, the continuous study of a very

fascinating subject. I again express my appreciation to Dr. Johnston for bringing this up. I had not planned to discuss it until Dr. Roper infuriated me—but since he did, I wanted to give my ideas and particularly wanted to compliment Dr. Johnston for doing this work. I hope he will continue it.

David Shoch,
Corresponding Secretary.

SWISS OPHTHALMOLOGICAL SOCIETY

September 27-29, 1957
Zurich, Switzerland

Fundus findings

Dr. F. Rintelen and Dr. F. Hegglin comprehensively surveyed the problem of hypertension from the viewpoints of the ophthalmologist and internist respectively.

Dr. A. Huber and Dr. B. Cagianut (Zurich) noted that the differential diagnosis of papilledema due to malignant hypertension and that due to choked disc may be difficult when the hypertension causes cerebral symptoms or when a basal tumor produces hypertension. The presence of angiospasm, arteriosclerosis, cotton-wool exudates, and an excessive diastolic pressure of the central retinal artery found with Bailliart's dynamometer would indicate hypertension as the causative factor.

Dr. R. Witmer and Dr. A. Schmid (Berne) observed a cholesterin crystal embolus in the superior temporal retinal artery which was movable and caused no functional disturbance.

Dr. G. R. Constam (Zurich) discussed diabetic retinopathy in relation to arteriosclerosis.

Dr. J. Babel and Dr. B. Rilliet (Geneva) concluded from a study of 320 cases that diabetic retinopathy led to blindness in two to four percent of cases. They found that the blood values of cholesterin and glycoprotein increased with the severity of the retinopathy.

Dr. J. F. Cuendet (Lausanne) studied 46 patients with brittle diabetes under treatment with a sulfamide derivative (Tolbutamide). The effect on the advent of retinopathy was no more favorable than with insulin.

Dr. Brückner (Basle) suggested for diabetic retinopathy the auxiliary administration of testosterone and methiscol.

Dr. M. Favre (Berne) reported two cases of hyaloid-retinal degeneration of a type intermediate to those described by Wagner and Kleinert respectively.

Dr. A. Franceschetti and Dr. S. Forni (Geneva) described three cases of tapetoretinal degeneration with a curious marblelike appearance in the fundus periphery.

Dr. H. Goldmann (Berne) found that foci of choroiditis that escape ophthalmoscopic examination may often be seen with the slitlamp.

Glaucoma

Dr. A. Bangerter (St. Gall) uses iridectomy for acute glaucoma, usually peripheral; iridencleisis for narrow-angle glaucoma; and cyclodialysis or the Elliot trephining operation for open-angle glaucoma.

Dr. M. Amsler and Dr. E. Landolt (Zurich) remarked that but 80 cases of essential atrophy of the iris have been recorded in the past half-century. Their case ended in terminal glaucoma that required enucleation.

Dr. E. Landolt (Zurich) examined six eyes enucleated for absolute glaucoma which were definitely consecutive to a pre-existing primary glaucoma. In all, the histologic picture corresponded to hemorrhagic glaucoma secondary to obliteration of the central retinal vein.

Dr. U. Nemetz (Vienna) discussed three cases of glaucomatous cataract following keratoplasty.

Dr. E. B. Streiff and Dr. L. Golay (Lausanne) examined a 13-month male infant affected with Lowe's syndrome, and

found glaucoma, nystagmus, and concomitant divergent strabismus. In the other nine reported cases, glaucoma was noted in five, cataract in eight. All were males.

Dr. T. Schmidt (Berne) described an improved Schiøtz tonometer, designed by Goldmann and manufactured by the Haag-Streit firm, which avoids parallax and gives constant findings.

Dr. H. Heinren, Dr. P. Luder, and Dr. A. Muller (Zurich) stressed that a reliable value for ocular rigidity can be obtained only by using the Goldmann applanation tonometer and that of Schiøtz. Rigidity diminishes as myopia increases. In a myope, affected by glaucoma, the tension noted by the Schiøtz tonometer is less than that which truly exists.

Clinical investigation

Dr. B. Glocker and Dr. K. Mittelholzer (Geneva) found a positive dye test for toxoplasmosis in 43 percent of 536 cases of uveitis and but 22.6 percent in a control series.

Dr. G. Saubermann (Basle) has abandoned the Ridley technique and now uses intracameral lenses. He is convinced that most of the reaction to the plastic is due to the Zephiran used for disinfection. After removal from Zephiran the implant should be placed in distilled water for one hour and then thoroughly rinsed.

Dr. F. Frankhauser and Dr. T. Schmidt (Berne) would distinguish two types of perimetry—static and kinetic.

Dr. H. Dubler and Dr. J. J. Scheidegger (Geneva) studied the aqueous with the method of immuno-electrophoresis developed by Scheidegger. In uveitis due to Reiter's syndrome, a fraction found in the serum (B-2 m) was absent from the aqueous.

Dr. W. Bamert (St. Gall) examined 225 cases of monocular convergent strabismus and found some hypofunction of the lateral rectus in two thirds of the amblyopic eyes.

Dr. R. Fessl (St. Gall) found numerous cases of amblyopia refractory to Bangerter's methods of treatment.

Virus diseases

Dr. G. Töndury (Zurich) has been interested in the fetal cataracts resulting from virus infection in the mother in the first trimester of pregnancy. His animal experiments show that epithelial organs, and especially the lens, are most sensitive to the pathologic action of the virus.

Dr. P. Dieterle and Dr. A. Schwarz (Geneva) discussed the pseudoretinitis pigmentosa that may follow rubeola, rubella, and antismallpox vaccination. The prognosis is usually good but in some cases a permanent visual loss ensues.

Dr. C. Stucchi and Dr. A. Vollenweider (Lausanne) cited a case of Behçet's disease in which the fundi showed a pseudoretinitis pigmentosa similar to that found with other virus diseases.

Case reports

Dr. E. Rosselet and Dr. E. Beck (Lausanne) discussed the syndrome of Mauriac, a complication of infantile diabetes characterized by dwarfism and hepatomegaly with accordion pleating. These infants usually die before the appearance of eye lesions, though they have seen two with diabetic cataract and one with retinopathy.

Dr. E. Bürki (Basle) added further details to his 1954 report of a case of multiple myeloma diagnosed then by the slitlamp study of the cornea. In 1955 electrophoretic study of the serum showed a significant increase of gamma globulin. A third sternal puncture in 1956 for the first time gave positive findings. The patient died of uremia and the diagnosis was confirmed by autopsy.

Dr. J. Nordmann and Dr. A. Burger (Strasbourg) discussed a case in which the clinical history suggested a choroidal metastasis of melanoma but histologic study demonstrated that the melanosarcoma of the eye was of the primary type.

Dr. R. Witmer and Dr. A. Schmid

(Berne) told of a patient with optic atrophy in one eye secondary to temporal arteritis who was seen just after he developed a thrombosis of the central retinal artery in the other eye. Fortunately, treatment with anticoagulants and vasodilators proved effective.

John D. Blum, Geneva,
Correspondent.

James E. Lebensohn, Chicago,
Translator.

NEW YORK SOCIETY FOR CLINICAL OPHTHALMOLOGY

May 6, 1957

DR. MAX CHAMLIN, *President*

ELECTROCYCLOGRAPHY

DR. JERRY HART JACOBSON, DR. G. PETER HALBERG, AND DR. HUNTER H. ROMAINE reported on electrical activity of the human eye during accommodation. It has been recently discovered by Buhlbring that it is possible to record potentials from contracting smooth muscle. It has been known for a period of time that when a mechanical transducer is attached to the ciliary body and the ciliary ganglion is stimulated electrically, a contraction is observed. Schubert recently reported a potential of a slow tonic nature in the human eye during accommodation.

In their study, the present authors used a number of human volunteers, placed in a device which prevents eye motion but allows for accommodative changes. This consists of a large silver mirror with a near fixation dark spot superimposed upon a distant one in such a manner as to eliminate motion when a shift is made from one to the other.

It is their finding that, when the human eye accommodates, there is a deflection of positive electricity of a character similar to the form of mechanical change found in animals on ciliary ganglion stimulation. Experiments in patients whose accommodation was paralyzed with cycloplegics produced similar potential changes.

ACUTE GLAUCOMA AND GENERAL SURGERY

DR. SAMUEL GARTNER AND DR. EDWIN BILLET pointed out that acute glaucoma occurs in the early postoperative period after general surgery with sufficient frequency to bring the problem to the attention of ophthalmologists, surgeons, and anesthesiologists. In the past year at Montefiore Hospital four such cases were diagnosed out of a total of 3,437 surgical cases operated under general and spinal anesthesia. In none of these four cases did the patient have any awareness of eye trouble before operation. It is possible that other cases occurred where transient acute episodes were missed by the surgeon.

The acute attacks of glaucoma followed excision of a polyp of the large bowel in one case, abdominoperineal resection of the rectum in one case, and cholecystectomy in two cases.

The responsible factors are considered to be atroprine and scopolamine in premedication, deep anesthesia with pupillary dilation, the excitement of hospitalization, the semidark room following surgery, and the predisposition of these patients to acute attacks by the anatomic structure of their eyes.

The symptoms of acute glaucoma are usually overlooked in the immediate postoperative period.

It is suggested that pilocarpine hydrochloride (two percent) be instilled routinely in the eyes of patients prior to general anesthesia, and during the early postoperative period, in an endeavor to prevent acute attacks of glaucoma.

FRACTIONIZED CYLINDER RETINOSCOPY

DR. ALFRED KESTENBAUM said that Lindner's known rotation test in cylinder retinoscopy allows exact determination of the axis of the cylinder. The axis is correct if a rotation of the axis five degrees to the right or to the

left results in a new astigmatism, the stronger meridian of which lies to the left or to the right, respectively, in analogy with Jackson's rules for the "cross-cylinder" method. If the cylinder glass is correct in strength, the angle between the "new astigmatism" and the correct axis of the glass measures 42.5 degrees or around 45 degrees. If the glass is too strong, the angle is larger than 45 degrees. If the glass is too weak, the angle is less than 45 degrees. Lindner stressed that this fact can be used for determination of the exact strength only in low degrees of astigmatism, since the deviation of the angle from 45 degrees depends on the relative size of the error. If +1.0D. is given instead of +0.5D. this means an error of 100 percent, but if +5.5D. is given instead of +5.0D. the error is only 10 percent, so that the deviation of the angle from 45 degrees is not noticeable.

This difficulty was overcome by the author by means of fractionization of the cylinder in the rotation test. At first the axis is exactly checked by the rotation test. Then the cylinder is subdivided into a larger stationary part and a small mobile part; for example, +5.0D. is divided into +4.0D. and +1.0D. The large part, +4.0D., is put into and kept in the correct axis; only the small part, +1.0 D., is rotated five degrees alternately to the right and to the left and the angle between the "new astigmatism" and the correct axis of the glass is observed. Since only +1.0D. is subject to the rotation, an error of +0.5D. up or down gives a strong, noticeable deviation of the angle from 45 degrees. If the angle is more than 45 degrees, the cylinder is too high (if + cylinders are used); if the angle is less than 45 degrees, the cylinder is too low; if the angle is about 45 degrees, the strength of the turned cylinder is correct; this turned cylinder is added to the stationary part.

This fractionization of the cylinder allows one, therefore, to check, in addition to the axis, the strength of the required cylinder by rotation tests and to substantiate the result of the usual retinoscopy.

Jesse M. Levitt,
Recording Secretary.

COLLEGE OF PHYSICIANS OF PHILADELPHIA

Section on Ophthalmology

November 21, 1957

Dr. I. S. Tassman, *Chairman*

Twentieth annual deSchweinitz lecture

Dr. I. S. Tassman (introduction):

With each succeeding year, there are fewer among us who had the good fortune to know Dr. George E. deSchweinitz.

In the prime of his life, he was tall in stature, attractive in appearance, and pleasant in his manner. One was instantly impressed with his commanding personality. He won many awards and honors for his contributions to the development of ophthalmology and was recognized as the Dean of American ophthalmology.

Dr. deSchweinitz was professor of ophthalmology at the University of Pennsylvania from 1902 to 1924, president of this College of Physicians in Philadelphia from 1910 to 1913, president of the American Medical Association in 1922, and a member of all of the ophthalmologic societies in this country and abroad. He was the type of man who would have been an outstanding success in any field of endeavor.

Dr. deSchweinitz died on August 22, 1938, at the age of 79 years. The first deSchweinitz Lecture was delivered on November 17, 1938, by Dr. Edward Jackson on "The development of ophthalmology in one lifetime"—a most appropriate title.

Tonight we will hear the 20th annual deSchweinitz Lecture to be delivered by Dr.

John H. Dunnington of New York who is well known to most of us.

Some modern concepts of ocular wound healing

Dr. John H. Dunnington: Although the complex problems of wound repair are far from solved, much progress is being made. The healing of epithelium depends upon its rapidity of spread and its tendency to line all open crevices until advancing epithelial edges meet. These characteristics influence not only normal healing but also have much to do with the production of epithelial invasion of the anterior chamber. The pathogenesis of this complication of cataract extraction is discussed.

Some recent investigations of the reactions of the corneal stroma during healing are reviewed. Radioautographs indicate synthesis of sulfated mucopolysaccharides at the wound edge 17 to 24 hours after operation. The invasion of polymorphonuclear leukocytes can be inhibited if soybean trypsin inhibitor or sodium salicylate is applied to the incision during the second postoperative hour. Corneal fibroblasts arise first from the transformation of corneal stromal cells along the edge of the incision and later from monocytes which migrate into the injured area. The changes in the noncellular elements of the stroma during the healing of a limbal incision are described. Abnormalities of stromal healing are manifest by broad scars and stromal overgrowths. The clinical importance of avoiding these complications is outlined.

The present trend of thought concerning the role of Descemet's membrane and endothelium in healing is that endothelial healing takes place by the transformation of proliferating corneal fibroblasts.

William E. Krewson, 3rd,
Clerk.

Ophthalmic Miniature

The Eye, the noblest and most beautiful organ of the body, through which the mind receives its chief stores of knowledge, and many of its purest streams of joy—and by aid of which are carried on all avocations that contribute to the happiness and improvement of society—seems to have been the only part abandoned to an almost total neglect.

Edward Reynolds,

An address at the dedication of the New Building of the Massachusetts Eye and Ear Infirmary, July 3, 1850. Published by C. C. P. Moody, Boston, page 8.

AMERICAN JOURNAL OF OPHTHALMOLOGY

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A PLASTIC APPROACH TO SURGERY*

CHARLES HEANLEY,† F.R.C.S.
London, England

There is no such thing as a "lucky" surgeon—"the wind and the waves are on the side of the ablest navigator." To be lucky is to know the rules and to know when to break them.

The first rule is that diagnosis should precede treatment, and to disregard this rule is "worse than a crime, it is a mistake." The word "diagnosis" is merely a verbal symbol to represent a living and changing phenomenon; diagnosis in its fullest sense is an understanding of the pathologic process present.

* This paper appeared in full in *The Lancet,* January 11, 1958, p. 94.

† Consultant plastic surgeon, The London Hospital, Whitechapel, E. 1.

The making of a diagnosis is a continuous process which does not cease with the start of the operation, but should always be one step ahead of the treatment. It is fatal to cling to an incorrect diagnosis; so, having made a diagnosis, do not stick to it. A plastic outlook is essential. Each case should be treated on its own merits, since no two cases are the same. The man with a rigid method and technique is dangerous: if he says that something is always so, he is either teaching or no surgeon. Since pathologic processes change, what was a correct diagnosis at one time may be wrong later. One should start an operation with a working diagnosis, together with a clear conception of the known facts and an open mind.

One learns quickly from one's mistakes, sometimes too quickly. It is nicer to learn from the mistakes of others. However, it is essential to be able to recognize a mistake, and it may be hard to realize that the result might have been better. Inquisitiveness is an essential attribute of a good surgeon, because inquisitiveness leads to an increase of knowledge; and to know all about a condition is to know its complications, and thus avoid them.

Do not ignore the opinion of the uninstructed; for rude minds sometimes contain pearls of wisdom, and discussion is always helpful, because what is dark when unspoken may become clear when discussed.

Although the diagnosis is correct, it may be difficult to decide on the treatment, because this so much depends on the prognosis. The knowledge of the prognosis of a disease and the assessment of the prognosis in the particular case is essential in deciding the treatment, especially in carcinoma.

So often the problem is not how to do the operation, but what operation to do, since it is the duty of the surgeon to prolong living in comfort, rather than to prolong the discomfort of dying. A clear conception of why one is operating at all, and why the particular operation has been decided upon, should always be kept in mind.

You should not do today in an ill patient what can be done tomorrow or later in a fit patient. Emergency operations should be avoided if possible: the acute abdomen, for instance, is a Pandora's box of mischief. To an already ill patient do not add the insult of a big operation; he needs a life-saving operation, and no more. Do not try to paint the lily, for a live weed is better than a dead lily.

* * *

The gentle handling of tissues is essential; this rule must not be broken. Avoid lethal surgery, whether it is a cell or the patient that dies. The more dead or dying cells left in an operation site, the slower the recovery. The heavy-handed surgeon who mauls the tissues, often with the aid of fierce instruments and retractors, is the one who complicates already elaborate techniques in a vain attempt to reduce the incidence of postoperative sepsis.

There must be no tension, either in the surgeon or in the tissues. Healing is quickest when the tissues lie adjacent to one another without tension or compression. Both these states, which are associated with tight stitching, upset the blood-supply to the healing edge. Sutures should be inserted in such a way that the blood-vessels in the loop of the stitch are not compressed, either at the time of stitching or later when the tissues swell from postoperative edema: otherwise more trauma is caused, with more edema and more compression. Whereas a ligature is for hemostasis, a suture is to approximate tissues until they heal, and a stitch that is inserted to perform both these functions is essentially bad. . . .

Each stitch must be inserted and tied as if it were the most important one. Since a successful operation depends on its weakest stitch, each one should have meticulous care. As is seen in skin suture, a badly placed stitch cannot be corrected by any number of rightly placed stitches, so it should be removed at once. If the stitches are loose and

soon removed, only the incision will be visible, and not the stitch marks. But stitches can be removed early only if there is no tension and a good blood-supply. . . .

By your scars will you be judged—the patient honors the surgeon if the scar is neat, for if the internal malady is cured, what is left save the scar by which to judge the result? A neat suture line may cover a multitude of sins. Incisions should be made in the direction of so-called Lange's lines, which for the most part lie in the flexion creases. If a circle is drawn on the skin, and the circle of tissue is removed, the resultant bare area is larger than the area removed, and is oval in shape, showing that tension, and unequal tension, exists in the skin. Therefore, if the incision is made at right angles to direction of the least tension a good scar results; if it is made in another direction there may be a contracture.

"I shall excise the scar, the whole scar, and nothing but the scar, so help me Hippocrates," is the initiate oath of the plastic surgeon. The resulting defect is sometimes surprisingly large—one removes a pennyworth of scar and gets a shillingsworth of bare area. This is due to the fact that, where the skin is supple, skin defects heal by contracture of the scar tissue drawing the edges together, rather than by epithelial growth.

* * *

Nature abhors a vacuum, and fills it with a bloodclot; and, as hematomas are almost synonymous with wound infection, subcutaneous dead spaces can be obliterated by end-on mattress sutures of nonabsorbable material with the loops tied over packs. Some sites are more prone to the formation of hematomas than others. Whereas, blood-vessels in the face usually stop bleeding naturally, those in the lower abdomen do not; so every miserable bleeding blood-vessel must be ligated. In plastic surgery, pressure dressings are used to press the graft against the recipient area and so prevent hematomas. The introduction of the crêpe bandage has been the greatest advance in skin-grafting in the 20th century. To control hemorrhages, pressure dressings should be left for four days. Six days shall you labor and the seventh is for the evacuation of hematomas!

Raw surfaces should not be left exposed, because they granulate and form scar tissue. Scar tissue is feminine in gender, being perverse at all times: it contracts when it should relax, as in strictures; and stretches when it should contract, as in hernia repairs. To say that a wound is granulating well is a contradiction in terms. . . .

The best dressing for any raw surface is a skin graft, and the thicker the graft the less the contracture. Repeated dilatations are the signposts of surgical failure, and pull-through methods of anastomosis are bad. . . .

Hippocrates said, "Do not operate on growths—it makes them worse"; the same rule holds today. One should operate on the normal tissues away from the growth, and not cut across carcinomatous tissue. For the surgeon, the way to hell is paved with short incisions; for the patient, the way to heaven is paved with small excisions in carcinoma surgery. Carcinomas are seldom removed completely, but the growth of those cells that are left behind is held in check by many factors. As the radiotherapist depends on the tissue reaction of the healthy patient for the treatment of carcinoma, so does the general surgeon. The cynic says that in every case of carcinoma of the breast, recurrences will develop provided the patient lives long enough. But the early recurrence appears on the patient who has made a slow postoperative recovery. Therefore, she should be kept as fit as possible, before, during, and after operation. Large operations should be avoided wherever possible, because they lessen the general health of the patient and so upset the hormonal control. At the site of successfully treated carcinomas, plastic operations should be avoided lest they liberate carcinoma cells from the scar tissue.

* * *

To be a quick operator is good, but speed must never be gained by the sacrifice of any

principles. Thou shalt not worship speed for its own sake, for it is a false god. A quick surgeon never hurries, for speed is not made by over-haste; when you drive a car, a high average speed depends not on how fast you go, but on how slowly you don't go. Delay is due to indecision, which results from incorrect diagnosis, faulty planning, and lack of experience. The repetitive, unproductive movement should be avoided, as in time-and-motion study: the hand that puts down the scalpel picks up the artery clip—in one movement. Time is saved by attention to many details, such as the position of lights, trolleys, and assistants, and by teamwork. Time is saved, therefore, by performing slow, deliberate, economical movements, never unnecessarily repeated. Thus, speed becomes the handmaiden of the surgeon and not his mistress.

THE CARE AND NUTRITION OF THE GUEST SPEAKER

Each year during the Academy meeting the International Association of Eye, Ear, Nose, and Throat Secretaries have dinner together and discuss the methods of improving their local meetings. From Malaya to Little Creek to Belgrade the same questions arise, not the least of which are, "Who can we have to speak who doesn't live too distant, speak too abstrusely or tediously, who has something new to report (not a theme on exophthalmos again!), and won't use the same topic as the guest speaker of last year or last month?" Some societies apparently seek out only professors, believing that if his talk is not well received at least it is more likely a reflection upon his institution rather than the program committee. Others may emphasize youth, while other groups prefer an affable extrovert—a delight at joint meetings with the ladies' auxiliary, but not always a stimulating speaker after extensive hospitality.

A companion group, The Coterie of Perennial Peripatetic Guest Speakers, does not yet meet, but if they did perhaps their sessions would be as interesting as the secretaries'. The various meeting rooms would be certain to be a provocative topic: the academies of medicine—frequently located in an older part of town with superb speaking facilities but dinner an austere repast in a basement dining room; the country club—much entertainment before dinner, a fine steak dinner—but usually no lectern and a tilting projector, ladies' night bingo competing in an adjoining room and transient members believing that the Greens Committee is meeting and departing after several baffled inspections of the audience; the hotel—the tepid pot roast and cold peas, and the inability to start until the union sends a projectionist; the university—milk shake and a hamburger in the student lounge and "we will begin as soon as the classroom is free—didn't know the seminar was meeting this semester"; the large convention hall—all business, with an audience ebbing and flowing like the tide, as some realize this is not the Georgian Room and the lower backache section, and others patiently reviewing their newspapers or college reunion plans as they await the next speaker.

The speakers, too, could exchange the stories which they use to fill in the minutes while a frantic search is made for an extension cord so that the projector can be connected. They could discuss the even better filler material required when the only projector bulb available burns out midway in the talk. Experienced speakers, too, could explain how to prepare slides so that a pointer is never required since it is likely that none will be available until exactly two minutes prior to conclusion when an ingenious member will find a meter stick, a broken putter or a window pull-down pole.

An important topic is the use of colored slides. Commonly the projector is of such low wattage that any extraneous light successfully prevents the image from being seen on the screen. This is frequently an urgent signal for one of the audience to turn off all the lights in the room, including the circuit for illuminating the lectern and sometimes the projector, too.

Experienced speakers could caution against betraying emotion in those communities in which the audiences follow a suburban commuting schedule. The novice may be somewhat disconcerted as a third of the group arises to leave precisely at nine o'clock to catch the last train to Upper Broomswitch. The tyro must learn also to appreciate that the reduction of illumination is a signal for the departure of a few, even in those areas having no commuters. The experienced speaker, of course, knows too well the problems of somnolence in busy practitioners who have arisen early, striven to compensate for a late arrival at the fellowship hour, and then had a heavy dinner. Simple charity probably demands that the speaker not be too insistent on full illumination between slides.

A final topic for guest speakers could be the delicate one of expenses. Now that the Internal Revenue Department demands strict accounting of travelling expenses, this is a detail that certain conscientious treasurers might well leave to federal control. Certainly a number of questions arise after the simple phase: "Just send a list of your expenses and the treasurer will send you a check." Is the treasurer in town or will the check arrive after he has returned from six months in the Near East? Does the society pay interest for the investment in travel expenses between the time of the trip and the payment of the expenses? Does the society permit the guest speaker to have his clothes pressed and shoes shined, or is simple austerity and rumpled bonhomie the order of the day? Possibly, the speakers might even resolve to urge their companion group, the secretaries, to have the check covering expenses given to the speaker while he is visiting their community. The size of the check will vary with a number of factors but it hardly seems proper to reimburse on the basis of the treasurer's opinion of the talk, as Clarence Day's father judged his preacher. If the treasurer would prepare the check for exactly the same amount he would deduct from his income tax if he took a journey of a similar distance on a business matter, in all probability he would acquire a reputation for unparalleled generosity. Thus the agenda for meetings of the Coterie of Peripatetic Perennial Guest Speakers could be most impressive in range and interest. Other topics will be considered in future communications.

LUX LECTOR, C.P.P.G.S.

THE 17TH CLINICAL MEETING OF THE WILMER RESIDENTS ASSOCIATION

The Wilmer Residents Association is composed of former House Officers and Fellows of The Wilmer Ophthalmological Institute of The Johns Hopkins Hospital. The first clinical meeting was held on May 12, 13, and 14, 1938. The 17th meeting was held at The Johns Hopkins Hospital on March 28, 27, and 29, 1958. Approximately 350 ophthalmologists from the United States and Canada registered during the meetings. Thirty papers were read during the three-day session.

The program was opened on Thursday morning by the present resident, Jack S. Gans, who spoke on "Some techniques in ophthalmic plastic surgery." Dr. Gans stressed the importance of a carefully placed pressure dressing and described the usefulness of a continuous locked running suture in skin closure. The second paper was by the director of The Wilmer Institute, A. E. Maumenee, who spoke on "A new concept of the pathogenesis of congenital glaucoma." Dr. Maumenee discussed the previous theories of the pathogenesis of congenital glaucoma and then presented evidence to show that an abnormal insertion of ciliary muscle fibers to the trabecular network in front of the scleral spur was the cause of congenital glaucoma. Frederick Verhoeff of Boston who was present in his usual role of dissenting minority stated that he thought that

this paper was a very important contribution.

Stewart M. Wolff spoke on "Traumatic glaucoma," and described a peculiar deformity in the chamber angle after contusion of the eye in three cases. A very interesting case of "Cortical blindness with partial recovery following acute cerebral hypoxia from cardiac arrest" was presented by William Hoyt, Frank B. Walsh, and Richard Lindenberg. James R. Duke and David Clark discussed "Tay-Sachs disease in the Negro race." A review of the literature had disclosed no previous reported cases of Tay-Sachs disease in the Negro race and the authors presented two such cases examined clinically and pathologically.

The last paper of the morning was by J. Lawton Smith who discussed "Raeder's paratrigeminal syndrome." Dr. Smith discussed the clinical findings in eight such cases seen at The Johns Hopkins Hospital during the past 25 years and stressed the importance of accommodation studies in its diagnosis and differentiation from Horner's syndrome.

The first paper on Thursday afternoon was "Observations in clinical electromyography," by Marvin L. Sears, Robert D. Teasdall, and Howard H. Stone. This was followed by a paper by A. D. Ruedemann, Jr., of Detroit, on "The electroretinogram in retinitis pigmentosa." Dr. Ruedemann presented 38 cases of retinitis pigmentosa in which the electroretinogram was extinguished in 30 but was present to a varying degree in the remaining eight cases.

Richard E. Hoover discussed the "Current status of contact lenses" and this was followed by "Keratoplasty in herpetic keratitis" by Thomas Carey and A. E. Maumenee. The authors discussed the results in approximately 70 cases in which keratoplasty had been carried out for herpetic keratitis. Charles W. Tillett of Charlotte, North Carolina, discussed "The use of visual aids in office practice." Louise Sloan, Marvin L. Sears, and Maria D. Jablonski presented a paper on the "Clinical significance of the AC/A ratio." This was presented as a preliminary study and especially stressed the effects of DFP on this ratio. The final paper of the day was "Surgical treatment of scleromalacia perforans" by Malcolm W. Bick of Springfield, Massachusetts. He presented one case in which a fascia lata graft was placed over the defect.

An informal oyster roast supper was held at the Alcazar on Thursday evening. Delicious Maryland oysters were served in every shape and form and a delightfully informal evening was enjoyed by everyone. The Wilmer Residents Staff ended the evening with a short skit which was enjoyed by all of the out-of-town ophthalmologists and by some of the Baltimore ophthalmologists.

The first paper on Friday morning was by J. Wallace McMeel whose title was "studies on corneal infection with Pesudomonas aeruginosa." This was a continuation of his work reported a year ago. Jack C. Cooper presented a paper on the "Eye movements associated with myoclonus." The next two papers by Kenneth T. Brown and Torsten N. Wiesel on the "Intraretinal recording of electroretinogram and single cell activity in the unopened cat eye" and "Analysis of receptive fields in the cat retina" were of great interest and importance.

Alan C. Woods read a paper on the question "Is heterochromic iridocyclitis a disease entity?" and presented evidence to show that it was not a definite entity. One of the most interesting papers of the meeting was by John M. McLean of New York on "Steroid prophylaxis in sympathetic ophthalmia." He presented a case of perforating injury in a young boy who developed sympathetic ophthalmia in spite of intensive prophylactic therapy with steroids. The morning session concluded with a paper by L. Harrell Pierce on "Some aspects of retinal detachment surgery."

In the afternoon Walter C. Peterson presented an "Evaluation of dichlorphenamide in glaucoma." This was followed by a paper

on the "Preliminary results in a glaucoma survey" by Carteret Lawrence. Jack S. Guyton and M. Wyatt Haisten of Detroit discussed "Iridencleisis: Technique and results obtained in 195 consecutive operations." Howard H. Stone and Marvin L. Sears of the present house staff presented a paper on "Ocular tension in experimental cross-circulation."

Samuel D. McPherson, Jr., and Dubose Egleston of Winston-Salem presented their results in a "Review of 106 cases of dacryocystorhinostomy." Arnall Patz discussed the production of "Experimental ocular tumors" in the mouse eye using methyl cholanthrene as a carcinogenic agent. The final paper of the afternoon was by Herman K. Goldberg who spoke on the topic, "The ophthalmologist looks at the reading problem."

On Saturday morning the first paper was by Laurence B. Senterfit who discussed the "Variations in the protein compositions of normal and cataractous human lenses." The protein compositions were determined using paper electrophoresis. Robert A. Schimek of New Orleans presented an "Experimental and clinical evaluation of Gelfilm" and reported three cases in which he had used Gelfilm inserts in cyclodialysis for glaucoma.

As has been the custom for a number of years the final presentation of the meeting was the neuro-ophthalmologic clinic conducted by Frank B. Walsh. A fascinating group of cases was presented and discussed, including a case of leontiasis ossea, Best's macular dystrophy, a family with familial myasthenia gravis, a case of pituitary tumor with a sixth nerve palsy, and an epidermoid tumor of the frontal lobe and chiasm.

At the close of the meeting, it was announced that the 18th clinical meeting will be held on March 19, 20, and 21, 1959.

Howard Naquin.

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* Section on Ophthalmology, College of Physicians of Philadelphia.

OBITUARY

WILLIAM ZENTMAYER
(1864-1958)

Dr. William Zentmayer, one of the most active and prominent members of this Section,* died on the evening of March 18, 1958, in his 94th year, during an attack of angina pectoris. This attack was secondary to a coronary thrombosis suffered one month earlier. Thus passed a distinguished, possibly unique, figure in ophthalmology—a man who, during his active career, was one of a galaxy of prominent ophthalmologists.

Dr. Zentmayer was born October 28, 1864, the son of Joseph and Katherine Zentmayer. His father, who emigrated from Germany, was an eminent optician and he undoubtedly influenced his son to study ophthalmology. Mr. Zentmayer etstablished his firm in Philadelphia in 1853 and soon won world-wide renown for his work and inventions on microscopes. The Franklin Institute awarded him the Elliott Cresson gold medal in 1874, the Centennial Exhibition in Philadelphia a second medal in 1876, and the Paris Exhibition a third medal in 1878 for his work on microscopes. During this time, there was not a maker of microscopes in the world who did not use some of his important inventions. A photographic lens that he conceived theoretically, and then made, was so radically original, that when it was first described, his European confreres called it incredible and an "American exaggeration." This cause a controversy which is detailed in various issues of *The Journal of the Franklin Institute* and *The Philadelphia Photographer*. He was among the first to limit his business in spectacles to perscriptions from ophthalmologists; and, as early as 1860, ground spherocylindric lenses. It was due to men like Dr. Zentmayer and his father that, during this period, Philadelphia was one of the greatest ophthalmologic and optical centers in the world.

Dr. Zentmayer's numerous contributions to literature were all carefully and thought-

fully executed, and he was frequently asked to discuss papers because of his knowledge of ophthalmic literature. He was chairman of the Section on Ophthalmology of the American Medical Association, 1916-17, and president of the American Ophthalmological Society, 1926-27. These positions and others he obtained through sheer merit, as he was not especially gregarious, though a witty and interesting conversationalist. He was a bachelor, and his main interests were ophthalmology, poetry, and music. He was an insatiable reader and had a remarkably retentive memory for things ophthalmologic, so that his knowledge of ophthalmic literature was second to none, as many an essayist can testify.

Dr. Zentmayer was a good surgeon and performed a greater variety of operations than any one I know, particularly on the extraocular muscles. He was an excellent teacher and his lecture notes were in great demand. In 1919, he helped organize the basic training course in ophthalmology in the Graduate School of the University of Pennsylvania. During his active career, he was considered "the oracle" at Wills Eye Hospital, where all sorts of questions were put to him. If he was unable to answer a question at the moment, one could be sure that he would have a very full explanation within a day or so, having consulted his well-stocked library in the meantime. His editorial training made him very critical and I know of no one who so frequently discovered ophthalmology spelled incorrectly. He was a keen clinician and remembered his patients remarkably, and woe betide the resident who forgot his orders or findings of the previous clinic day. His forte, however, was ophthalmoscopy, and, curled up on a stool in the dark-room of the old Wills Hospital, he astounded students and ophthalmologists alike by his ability to determine the refraction of an eye with his insignificant-looking little reflecting light ophthalmoscope.

Dr. Zentmayer served as an editor of the *Archives of Ophthalmology* from 1930, when it was taken over by the American Medical Association, up to the time of his death, having previously served as associate editor. He enjoyed the friendship and respect of Dr. Arnold Knapp, who had edited and published this periodical at great expense to himself prior to this transfer. Dr. Zentmayer also served as editor of *The Transactions* of the College of Physicians of Philadephia from 1899 to 1913, and was very proud of his portrait by Daniel Garber, presented to that institution by an international group of ophthalmologists in 1940. He was president of the Medical Alumni Society of the University of Pennsylvania in 1922-23 and was guest of honor at the annual meeting of the American Medical Association in 1941.

The St. Louis Society for the Blind awarded him the Leslie Dana Medal in 1945 for "outstanding achievements in the prevention of blindness and the conservation of vision"; and in 1948, he was awarded the Lucian Howe Medal by the American Ophthalmological Society "in recognition of his distinguished service to ophthalmogy."

Dr. Zentmayer began his career as chief of the Eye, Ear, Nose, and Throat Out-patient Department of the Children's Hospital in 1887. He also served at the Glen Mills School, St. Mary's Hospital, St. Agnes Hospital, and The Wills Eye Hospital, where he became attending surgeon in 1901. He resigned from this position at the age of 65 years of his own volition and retired from active practice February 2, 1951—largely, I believe, because of a macular choroiditis. Fortunately, he was able to read with one eye up to the time of his death, and thus continue his interest in ophthalmology.

Dr. Zentmayer was very regular in his habits and punctilious in the amenities of his scientific and social life. During his active career, he smoked two cigarettes daily, one when he finished his clinic and one after dinner. He rarely, if ever, missed a section meeting of the College of Physicians of Philadelphia and I doubt if he missed many meetings of the American Ophthalmological Society. He often decried the present day

need of guest speakers at this section. He walked daily and knew all of the walking paths at Hot Springs, Virginia, and his favorite summer resort, Lake Minnewaska, where he would cover as much as 15 miles over mountainous terrain, hopping along like a bird. As a younger man, he bicycled and was very proud of his "century" or 100 mile ride from Philadelphia to Atlantic City and return on a warm summer day, and particularly of achieving this without wilting his high starched wing collar! Indeed, he belied the oft-heard athletic adage that the "legs go first." I doubt if he enjoyed anything more than the various ophthalmologic meetings, especially those of the Section on Ophthalmology, College of Physicians of Philadelphia, and the American Ophthalmological Society, and one could fill a book with his numerous and interesting anecdotes of the various members.

At the time of his death, he was consulting surgeon to the Wills Eye Hospital, Emeritus Professor of Ophthalmology at the Graduate School of the University of Pennsylvania, an editor of the *Archives of Ophthalmology*, and a life member of the National Society for the Prevention of Blindness. He lived at 265 Forrest Road, Merion Station, Pennsylvania, and is survived by a sister, Mary.

Warren S. Reese.

BOOK REVIEWS

STRABISMUS OPHTHALMIC SYMPOSIUM II. Edited by James H. Allen, M.D. Saint Louis, C. V. Mosby Company, 1958. 541 pages, 251 illustrations, index. Price: $16.00.

The first *Strabismus Ophthalmic Symposium*, edited by Dr. Allen and published by Mosby in 1950, proved to be very popular. Its contents represented the teaching of many American experts in the field of ocular motility. These were F. H. Adler, H. W. Brown, H. M. Burian, F. D. Costenbader, W. H. Fink, G. P. Guibor, C. S. O'Brien, R. G. Scobee, K. C. Swan, and the old maestro no longer with us, alas, Walter B. Lancaster.

The second symposium, just published, has chapters written by the above, with the exception of O'Brien, Scobee, and Lancaster. Their places are taken by Philip Knapp, the son of the late Arnold Knapp and a rising star. The new volume is appropriately dedicated to the memory of W. B. Lancaster and R. G. Scobee, and, in addition, two of Dr. Lancaster's papers are reprinted in the appendix of this volume.

It is gratifying to see the advances that have occurred in these eight years. Ophthalmologists in the last 15 years or so have become more or less familiar with the work of these men. They have brought much order out of the chaos surrounding our understandign of neuromuscular anomalies of the eyes. The contributions of these American "muscle men" have had world-wide influence. It is a pleasure, therefore, to have in our hands a well-printed and well-illustrated book, each chapter of which represents the latest thoughts of these experts, each of whom is a specialist within a specialty.

Fink's chapter is on the anatomy of the extrinsic muscles of the eye, as one would expect. It is most rewarding. Swan writes on the nature of normal binocular vision, the interrelationship of the sensory and motor components of binocular vision, the blind-spot mechanism in strabismus, and recession under Tenon's capsule.

Adler discusses the voluntary mechanism for eye movements and the involuntary or reflex mechanisms. Burian's chapters include the etiology of heterophoria and heterotropia, normal and anomalous correspondence, and the diagnosis of neuromuscular anomalies of the eyes.

Brown's contribution includes the use of the cover test, congenital structural anomalies of the muscles, and surgery on the oblique muscles. Guibor's chapters are on some uses of ophthalmic prisms, clinical application of

neurology, and the use of atropine in the treatment of strabismus.

Costenbader discusses the principles and techniques of nonsurgical treatment and the clinical course and management of esotropia. Finally, Philip Knapp discusses divergent deviations and their treatment and gives us a sound chapter on the surgical treatment of strabismus.

One of the most valuable parts of the book is a verbatim recording of an interesting round-table discussion in which Breinin, expert on electromyographic studies of the ocular muscles, takes part. This is delightfully informal and full of meat. It brings out very nicely the fact that even these top-flight ophthalmologists don't always agree and that there is still much to be learned. But we have indeed come a long way in the last two decades; thanks to these and other investigators.

The book is well edited, co-ordinating the different literary styles of the authors nicely and fluently. But, I warn you that, even so, it is difficult reading, for the subject is complex, as we all know, and at times the terminology is confusing, too. You will have to stop many times and go back over what you have just read. But this is most rewarding. I wish I had had this book when I was a resident 35 years ago. EHEU.

Derrick Vail.

APPLIED PHYSIOLOGY OF THE EYE. By H. Willoughby, M.D., assisted by T. Keith Lyle, M.D. London, 1958, Bailliere, Tindall, and Cox. (Williams and Wilkins Co., Baltimore, exclusive U. S. agents.) 314 pages and index. Price: $9.00.

The book consists of 22 chapters covering the subjects usually considered in a textbook on the physiology of the eye. According to the author it was intended as a book "to help those who are working for higher examinations in ophthalmology."

The book is very disappointing and would seem inadequate for its intended purpose. For example, only two pages are devoted to the physiology of the retina, the remaining eight pages of the chapter containing brief clinical descriptions of the various types of retinopathies. A contemporary physiology of the eye, on the other hand, devotes 15 pages to the physiology of the retina alone.

Similarly, in other important phases of physiology, little space is devoted to the subject; for example, six pages on the lens, four and a half pages on the vitreous, and six and a half pages on the aqueous. A great deal of the space of the book is devoted to very brief descriptions of some of the clinical affections of the eye.

The book has a place as a simple text for medical students or general practitioners who may be interested in the physiology of the eye. It cannot be recommended for the serious student in ophthalmology.

Frederick C. Cordes.

GESCHICHTE DER DEUTSCHEN OPHTHALMOLOGISCHEN GESELLSCHAFT. By Albert Esser. Munich, J. F. Bergmann, 1957. 84 pages, 3 illustrations. Price: not listed.

The *deutsche ophthalmologische Gesellschaft* commissioned Albert Esser to write its history in commemoration of its centennial. Although this society was formally inagurated on only September 5, 1863, it really was conceived during an informal meeting that took place in Heidelberg from September 3 to September 5, 1857. This meeting had been planned as a friendly gathering by v. Graefe and Donders to precede the first International Congress of Ophthalmology in Brussels and was attended by 12 ophthalmologists. Donders who had suggested Heidelberg as the meeting place in preference to Berlin, Graefe's choice, did not participate. His suggestion proved to be sound: with the exception of the meetings in Jena (1922), Leipzig (1932), Dresden (1940), and Munich (1950) Heidelberg has remained the permanent place of the meetings up to this day. The first small group found the Hotel

Schrieder an adequate place. Soon the ever-increasing number of participants made it necessary to move to larger quarters like the University Eye Clinic, the auditorium of the university, or some of the larger municipal buildings.

From the very beginning, Graefe was the moving spirit of the organization. It was he who had conceived the idea and had written the by-laws. His overwhelming personality dominated the meetings during his lifetime and has left its imprint to this day.

With its foundation in 1863, the *ophthalmologische Gesellschaft* (the name was changed to *deutsche ophthalmologische Gesellschaft* only in 1920) not only became the first society of any medical speciality in Germany but also the oldest ophthalmologic organization anywhere, preceding the American Ophthalmological Society founded in 1864. Its transactions were published in the *Monatsblaetter fuer Augenheilkunde*, published for the first time also in 1863.

A committee of six (one year later increased to eight)was in charge of all arrangements until the society became incorporated in 1902, when it became necessary to elect a president. The first man so honored was Leber. His successors were Uhthoff, Axenfeld, Wagenmann, Loehlein, Wessely, Lindner, and Thiel. In the entire history of the society, there were only three secretaries: Wilhelm Hess, Wagenmann, and Engelking who is still in office.

Although it had originally been planned to hold yearly meetings, it soon proved to be expedient to omit those years which coincided with an international congress. Exceptions were the seventh International Congress held simultaneously with the *ophthalmologische Gesellschaft* in Heidelberg in 1888 and the 1950 meeting, the year of the International Congress in London. In time of war and during post-war periods, meetings had to be dispensed with quite frequently.

Graefe had stressed the international character of the organization from the very beginning. Thanks to the pre-eminence of Donders, this outlook was very much in evidence during the latter's lifetime. The large number of non-German members, amounting at times to 38 percent, is eloquent testimony of the same spirit during the entire history of the group. The change in name from *"ophthalmologische Gesellschaft"* to *"deutsche ophthalmologische Gesellschaft"* should not be interpreted as a deviation from this ideal; as Uhthoff expressed it in 1925, the change was adopted as a form of protest against attempts to "boycott and isolate German research and science."

The Graefe Medal, the highest award of the society, was bestowed for the first time on Helmholtz in 1886. The original plan had been to make a presentation of the award every 10 years; this plan was not strictly adhered to. Recipients of the medal were Leber, Hering, and Carl Hess (the official ceremony took place after his death). The inflation that followed Warld War I exhausted the funds of the society and seemed to jeopardize the future of the Greafe Medal. Barkan's generous donation of 250 dollars came to the rescue. Further awards were made to Gullstrand, Gonin (posth.), and Thiel.

While the Graefe Medal definitely has an international character, the Graefe Prize is more national in character since it is awarded to the author of an article published in German in the *Archiv fuer Ophthalmologie*. This prize was originated by Robert v. Welz, a disciple and friend of Graefe, who made the fund available in 1873.

Of interest is the Dr. Joseph Schneider-von Welz donation made by Schneider of Milwaukee in 1913. In counterdistinction to the Graefe Prize which is awarded to the author of the best paper published during the preceding three years, this prize was granted to contestants who submitted an original paper with the sole purpose of obtaining this award. Oddly enough, the only applicant was Lindner in 1920. As a result of the post-war inflation this as well as other

prizes had to be discontinued. At present there remains the Axenfeld Memorial Foundation and the Uhthoff Foundation in addition to the Graefe Prize.

Members who have earned special consideration for their devotion to the society may be made honorary members; honorary members are Wagemann, Loehlein, Stock, and Comberg.

It is a pleasure to peruse this small volume. Not only does it re-emphasize Graefe's important role in the history of ophthalmology but also his close association with men like Donders, Bowman, and Arlt on a truely international level. His example in this respect has created a pattern for the international relation among ophthalmologists that is predominant to this day.

Stefan Van Wien.

THE BLOOD CIRCULATION IN THE RETINA (Der Blutkreislauf der Netzhaut). By K. Thuránszky (with an introduction by Prof. Dr. F. Kukán). Budapest, Adadémai Kiadó, 1957. 140 pages, 63 illustrations, bibliography. Price: not listed.

K. Thuránszky, a pharmacologist, selected the cat for his investigations because its retinal blood circulation is rather similar to that in man. Though he concerns himself in his experiments mostly with physiologic and pharmacologic aspects, his conclusions on controversial subjects as well as some highly original research concepts are equally important to the ophthalmologist. The ingenious experimental arrangements may be quite suitable as the basis for related problems in the field of ophthalmology.

Careful immobilization of the animal and its globe and general anesthesia permit removal of the cornea, the lens, and even the vitreous without distortion of the retina. The objective of a Leitz Ultropak microscope (with vertical illuminator) is provided with a cone-shaped glass rod that can be immersed into the vitreous or, if the latter has been removed, into normal saline or aqueous filling the cavum oculi, thus eliminating surface reflexes.

The blood pressure is checked with the equipment designed by J. Balassy: it registers graphically the fluctuations of the blood column in a polyethylene tube introduced into the femoral artery. These fluctuations cause an induction current which is amplified to actuate an electromagnetic registration mechanism. The author uses a clever method to stabilize the blood pressure in the experimental animal with a compressor. At the same time, this arrangement allows for a lowering of the blood pressure. The resulting slowdown of the circulation in the retinal vessels permits the visualization of individual blood cells. If desired, the circulation in the retinal vessels can be slowed down without reducing the blood pressure by applying clamps to both carotid arteries. An electronic flash bulb permits exposures of 1/5,000 sec. under magnifications up to ×150. For histologic examination, the retina is quick-frozen by a mixture of carbon dioxide snow and ether at the desired moment.

In a special chapter on the anatomy of the retina of the cat, the author refers to some of his previous investigations that enabled him to prove that, in addition to the narrow capillaries in the central area, there are wider and shorter capillaries in a three to four-mm. zone of the extreme retinal periphery that allow for a blood flow from the arterial to the venous side more or less independent of the central capillary circulation. He calls these vessels "shunt capillaries." He was able to demonstrate conclusively that the caliber of the central capillaries varies to such an extent that, at any given time, only a limited number were wide enough to allow the passage of erythrocytes, whereas all other central capillaries contained only plasma. The width of the capillaries varied continuously depending on the retinal metabolism in a given area. He believes that, on the basis of these findings, he can repudiate the idea of the so-called "vasa serosa" described by Cohnheim and Zuntz.

In his investigation of the sludge phenomenon, Thuránszky was able to prove that it is not characteristic of any specific pathologic condition but can be produced by lowering the blood pressure (either by mechanical or pharmacologic means) and by slowing up the circulation. It is a reversible process.

Stimulation of either the ipsilateral or contralateral peripheral sympathetic nerves increases the speed of circulation without changing the caliber of the retinal arteries. Adrenalin acts as a vasoconstrictor on the retinal vessels. If applied locally this effect takes place only with concentrations that are out of question for therapeutic purposes. Intravenous application causes passive vasodilation of the larger retinal vessels secondary to an increase in blood pressure. Acetylcholine has the effect of a dilator only after the fall in blood pressure caused by this substance has ceased.

There follows a discussion of the actions of other pharmacologic agents. In summarizing these actions, it can be stated that they may result in vasoconstriction or vasodilation as well as an increase or decrease in the speed of the retinal circulation. These results depend on the amount and the form of application of these agents as well as on their action on the systemic pressure.

Thuránszky is fully aware of the fact that his investigations were carried out under unphysiologic conditions: the opening of the globes eliminated the intraocular pressure as one of the important factors, although this elimination could be only of quantitative but not of qualitative significance. This reasoning probably is correct for physiologic and pharmacologic research. It is conceivable that the usefulness of this method might be limited for certain investigations of ophthalmologic interest, for instance in the field of glaucoma.

A wealth of material is to be found in this small volume. The experiments were conceived and carried out with tremendous ingeniousness and resourcefulness, with no detail left to chance. It is fortunate that the book has been published in German rather than the author's mother tongue. This makes it accessible to a much larger number of researchers. The excellent translation is by A. Faragó. The breathtaking photographs are splendidly reproduced.

Stefan Van Wien.

BIOMICROSCOPY OF THE VITREOUS AND THE FUNDUS. A. Busacca, G. Goldmann, and S. Schiff-Wertheimer. Report presented before the Société Française d'Ophtalmologie, May 13, 1957. Paris, 1957. Masson et Cie. 382 pages, 144 figures, 40 color plates. Price: not listed.

It is a great pleasure to write this review because I can with a clear conscience shout high praise. The text in which this exhaustive monographic discussion is written is clear, lucid, and perspicuous without a hint of turgidity or pendantry. The 40 plates are beautifully printed in color on glossy paper and are reproductions of more than 150 excellent informative drawings.

Some introductory remarks and the first chapter which deals with a history of the slitlamp microscopy of the posterior segment of the eye are the work of Schiff-Wertheimer. In the second chapter Goldmann describes the technique of examination of the posterior segment and in the third he provides an extensive analysis of the geometric optics of concave and convex accessory lenses and the contact glass with three mirrors. He also discusses the effect of spheric aberration of the optic system on the image of the slit as seen with the binocular microscope.

The fourth chapter by Busacca is devoted to the anterior portion of the vitreous. He describes the method of examination, important anatomic minutiae, the biomicroscopy of the normal vitreous in the region of its patellar fossa, anterior vitreous detachment, modifications of the anterior vitreous after extracapsular extraction of the lens and also

after intracapsular extraction, and hemorrhages and exudates in the vitreous.

The posterior vitreous is dealt with by Goldmann in the fifth chapter. He describes the special technique of examination, the normal biomicroscopic image of the vitreous, its optically separable bands, Cloquet's canal, and biomicroscopic aspects of posterior detachment of the vitreous. Pathogenesis and significance of posterior detachment and other pathologic alterations of the vitreous are described extensively and in detail.

In the sixth chapter, on the periphery of the fundus, Goldmann describes the technique of examination, some preliminary anatomic data, aspects of the periphery of the normal fundus, and such abnormalities of this region as malformations, degenerative changes, and the results of inflammation and hemorrhage.

In the long seventh chapter Busacca discusses the biomicroscopy of the normal fundus, describes the generalities of retinal lesions, and finally those of the macular region and the choroid. Schiff-Wertheimer similarly discusses the biomicroscopy of retinal detachment in the eighth chapter and cystic edema of the retina in the ninth.

The last three chapters are all by Goldmann; the 10th is devoted to intraocular neoplasms and the 11th to the optic disc. The 12th chapter consists of brief notes by Goldmann on surgery of the posterior segment of the eye under guidance of slitlamp observation, a procedure which is in its infancy but is, in the opinion of the author, completely assured of a bright future.

F. H. Haessler.

PRINCIPLES OF PERCEPTION. By S. Howard Bartley, Ph.D. New York, Harper & Bros., 1958. 482 pages, selected bibliography, index. Price: $6.50.

This college text is not simply a feat of scholarly assimilation in which Bartley mirrors the views of other writers. No, it is quite otherwise—a unique, unified work in which every aspect of perception is treated with insight and incisive thought. Extreme emphasis is placed on clear terminology. Wherever possible he uses a separate word for stimulus and *response*, as exemplified by —form, *shape;* photic pulse, *flash;* target intensity, *brightness*. He holds that in technical language a word must have no synonyms, and so differentiates carefully the shades of meaning in the following words applied to the human being: individual, subject, observer, organism, person. The goal of this study is enlightenment on how the human organism achieves personality.

Perception involves the interaction of two domains: the stimulus, originating in the physical world, that provides the energy to excite sense organs; and the response, which depends on the experience of the organism, its awareness of itself and of objects, and of the relations between them. Perception is subject to development and can be evoked by an object or the name of the object (symbolism).

About one third of the book details the results of psychosensory research on vision, in which Bartley has made many personal contributions. He showed, for instance, that under certain conditions a single photic pulse produced a pair of flashes, due to the different chronaxie of simultaneously activated rods and cones. The peripheral origin was proved by demonstrating that under similar conditions the optic nerve discharge of the rabbit likewise gave a double response. The classical viewpoint considers the seeing process as primarily a matter of optics, anatomy, and neural reflexes. Bartley would go beyond this, he maintains that vision is a learned achievement and very definitely believes in "visual training."

This work will reward the ophthalmologist with many new facts and a broader vista. The greatest of the books written by the great ophthalmologist, Sir John Parsons, was *An Introduction to the Theory of Perception* (1927).

James E. Lebensohn.

L'HÉRÉDITÉ EN OPHTALMOLOGIE. By Jules François. Bulletin of the Société Belge d'Ophtalmologie No. 118, Vol. 1. Brussels, Imprimerie Medicale et Scientifique, 1958. 298 pages, 63 illustrations. Price: Not listed.

The author, well known in this country, is an extraordinarily voluminous contributor of numerous scientific articles covering all phases, experimental and clinical, of ophthalmology. He is a sort of universal ophthalmologist of which there are very few. His contributions on all subjects set a high standard and have been most valuable. He is amazingly energetic and enthusiastic, this "gent from Ghent," as Al Reese once called him.

He was wisely chosen by the committee of the Belgian Society of Ophthalmology to prepare a report on heredity in ophthalmology. "J'ai accepté avec enthousiasme, car les problèmes génétique m'ont toujours beaucoup intéressé," he said.

This enthusiasm and interest in the subject is readily seen throughout the pages of his monograph. The subject is vast, the reference literature scattered in many nonophthalmologic journals, and some of it is inaccessible. In spite of these problems, Professor François has covered the ground surprisingly well. To do a complete job would require years of time and many volumes.

The monograph, therefore, has curtailed material, of course, but it is more than a summary or outline, and is a real addition to our knowledge.

Heredity and genetics have been more or less a neglected field in ophthalmology, in spite of the fact that many of our hereditary disorders have ocular manifestations, even in the puzzling "formes frustes" of these disorders. Ophthalmologists, therefore, who know this subject are in a position to advise with authority the physician in charge of the case and the parents of the affected offspring.

It would be a service to the ophthalmologist who does not read French, to have this splendid little monograph translated into English for us.

Derrick Vail.

ABSTRACT DEPARTMENT

EDITED BY DR. F. HERBERT HAESSLER

Abstracts are classified under the divisions listed below. It must be remembered that any given paper may belong to several divisions of ophthalmology, although here it is mentioned only in one. Not all of the headings will necessarily be found in any one issue of the Journal.

CLASSIFICATION

1. Anatomy, embryology, and comparative ophthalmology
2. General pathology, bacteriology, immunology
3. Vegetative physiology, biochemistry, pharmacology, toxicology
4. Physiologic optics, refraction, color vision
5. Diagnosis and therapy
6. Ocular motility
7. Conjunctiva, cornea, sclera
8. Uvea, sympathetic disease, aqueous
9. Glaucoma and ocular tension
10. Crystalline lens
11. Retina and vitreous
12. Optic nerve and chiasm
13. Neuro-ophthalmology
14. Eyeball, orbit, sinuses
15. Eyelids, lacrimal apparatus
16. Tumors
17. Injuries
18. Systemic disease and parasites
19. Congenital deformities, heredity
20. Hygiene, sociology, education, and history

5

DIAGNOSIS AND THERAPY

Costi, C. **Our experience with hyaluronidase.** Arch. Soc. oftal. hispano-am. **18**:187-190, March, 1958.

The literature on the use of hyaluronidase in ophthalmology is briefly reviewed, and the author's personal experiences reported. Its value in operations on the lids, the lacrimal passages and retinal detachment is no longer controversial. The author found a marked instability of the ocular tension following the injection of hyaluronidase, and for this reason he abandoned its use in glaucoma surgery. Retrobulbar injections of hyaluronidase were found effective in clearing vitreous opacities following uveitis or retinal detachment surgery. It is also effective in the restoration of postoperative flat anterior chambers as well as in promoting absorption of vitreous hemorrhages, provided that it is administered before an insoluble coagulum has formed. In a large material in the Institute of Medical Pathology the author found that in exophthalmos caused by a thyroid disturbance the retrobulbar injections of hyaluronidase were totally ineffective, while in malignant exophthalmos caused by a pituitary disturbance the exophthalmos was reduced by three or four mm. after two injections. The difference in the effect of hyaluronidase in the two types of exophthalmos was so marked that the author advocated the procedure for diagnostic purposes. The contraindications to the use of hyaluronidase are infectious foci, virus infections and malignant tumors; in these cases injections of hyaluronidase may lead to a dissemination of the disease, with grave consequences. Ray K. Daily.

Hambresin, L. and de Muellenaere, H. **Thrombosis of the cavernous sinus. A therapeutic problem.** Bull. Soc. belge d'opht, **117**:482-488, 1957.

In a 13-year-old boy thrombosis of the cavernous sinus followed a furuncle of the nose. The initial treatment with penicillin and terramycin did not prevent the progress of the disease. Cultures revealed Staphylococcus aureus as the offending organism, sensitive to terramycin, aureomycin, tetracycline and chloromycetin. Cultures from the infected sinus disclosed a mixed infection with Bacillus coli, sensitive to chloromycetin and tetracyline. Under specific therapy the child recovered completely. The importance of antibiograms in the presence of drug-resistant germs and the importance of anticoagulants inspite of their inherent dangers are

discussed. The beneficial results of the treatment possible now is compared with the disastrous outcome of sinus thrombosis only a few years ago.

Alice R. Deutsch.

Karpe, G., Kornerup, T. and Wulfing, B. **The clinical electroretinogram. VIII. The electroretinogram in diabetic retinopathy.** Acta ophth. 36:281-291, 1958.

In 74 diabetics with various types of retinopathy the ERG did not reveal specific characteristics. The relatively low b-potential corresponded to the extent and severity of the retinal damage. In some cases with only slight retinopathy the ERG showed signs typical of venous stasis. In diabetics with cataract the ERG may give valuable information about the state of the retina. (6 tables, 39 references)

John J. Stern.

Kluyskens, J., Titeca, J. and Popowycz. **Electroencephalographic control of the borderlines of functional disturbances of the visual fields.** Bull. Soc. belge d'opht. 177:445-468, 1957.

Previous studies on the interrelationship of the EEG and visual field borders had revealed that the appearance of a visual stimulus in the peripheral field caused an interruption in the alpha rhythm of the occipital lead of the EEG. This halt coincided invariably with the correct borderline of the peripheral fields, both in normal cases and in pathologic constrictions of various etiology. The present study had been undertaken to explore these connections by comparing the subjective admitted visual stimulation with the objective finding, the interruption in the EEG; 53 patients with severe functional field restrictions were examined. This series included 21 patients with a post-concussion syndrome, 12 with psychoneurosis and 10 malingerers. In every single case the R.A. (interruption in alpha-rhythm) referred to normal fields, while the subjective field showed more or less pronounced concentric construction. In spite of the fact that the interruption in the EEG is not perfect proof of visual perception, the discrepancies between objective and subjective findings aroused suspicion of wilful aggravation of perceptible symptoms. Modifications in the techniques of the tests presently used are described. Ten selected and typical case histories are reported in greater detail and copies of the individual EEG are included. The accuracy of an examination in complete darkness without a distinct fixation point and the possibility of cerebral stimulation of a different nature are considered as temporary disadvantages of an otherwise interesting and promising neurophysiologic investigation. 10 figures, 5 references)

Alice R. Deutsch.

Kornerup, Tore. **The clinical significance of the determination of the LK-value with special reference to perforating eye injuries.** Acta ophth. 36:292-303, 1958.

The LK-value is the wave length of light at which the retinal vessels and the fundus reflect the same amount of light. Its determination is a kind of oxymetry in reflected light. The LK-value was determined in 31 cases of iridocyclitis and 86 cases of perforating eye injuries with a modified method, using the visible ciliary vessels. An agreement between the LK-values and the usual clinical signs was found in about 80 percent of the determinations. In iridocylitis the reading of the LK-value is a help in studying the effect of treatment. In perforating injuries it is a quantitative method for following the course of healing and it may have prognostic significance because changes in LK-values may precede changes in the clinical signs. Fluctuating LK-values are found with a nonspecific chronic uveitis. In sympathetic ophthalmia the LK-values were high and fluctuating. Low or decreasing LK-values occur in small inflammatory foci at the limbus, in the

ciliary body or the iris. (2 figures, 1 table, 8 references) John J. Stern.

Larsson, S., Norman, O. and Hedbys, B. **Localization and extraction of intraocular foreign bodies by the method of Larsson.** Acta ophth. **36**:345-355, 1958.

Larsson's method for open bone-free roentgen examination for localization of intraocular foreign bodies was used in 40 cases with successful extraction in 28. Episcleral indicators were used. The method proved particularly useful in foreign bodies near the wall of the eyeball. (4 figures, 2 tables, 14 references)
John J. Stern.

Liegl, Otmar. **Trypsine treatment in ophthalmology.** Klin. Monatsbl. f. Augenh. **132**:486-497, 1958.

Trypsine was given to 61 patients; it was most frequently administered in an eye bath but sometimes injected subconjunctivally or intramuscularly. The results were especially good in 21 patients with dendritic keratitis. (30 references)
Frederick C. Blodi.

Ogg, A. J. **Examination of the eye with infra-red radiation.** Brit. J. Ophth. **42**:306-310, May, 1958.

In 1933 the first photographs were published of the anterior chamber of an eye made through an opaque cornea with an infra-red film. Since then several infra-red converter tubes have been described for viewing the eye directly. The British Army-type tube has several advantages over the others in that it is less expensive and is easily available. This tube can be attached to the arm of the Zeiss slitlamp for routine clinical use. (4 figures, 13 references) Morris Kaplan.

Olivares, M. **Photocoagulation.** Arch. chil. de oftal. **39**:113-117, July-Dec., 1957.

The author describes the fundamentals of the photocautery which Meyer-Schickerath devised for coagulation of lesions in the eyeground and treatment of retinal detachment.

The light source used must be very potent; the voltaic arch of Beck, which produces six thousand degrees kelvin is used.

Clinically it has been shown that in order to obtain areas of coagulation as localized and controlled as is desirable, the exposure to this light should not be more than four seconds.

Only tissues which absorb light can be benefited by the transformation of light into heat and therefore the retina will not show coagulation areas, unless it is in contact with the choroid or separated at the most two diopters. By the same reason, tumors of white color will not be affected by this treatment, whereas melanomas and like tumors are highly susceptible.

The photocautery has also been used to produce artificial pupils in pupillary seclusion following cataract surgery. (1 figure)
Walter Mayer.

Pinto Grote, C. **General anesthesia in ophthalmology.** Arch. Soc. oftal. hispanoam. **18**:255-263, March, 1958.

This is a general discussion of the indications of general anesthesia in ophthalmology, the various types of general anesthesia suitable for ophthalmic surgery, the techniques for various ages of patients, the premedication, and the risks involved. Unless there are specific indications, the author does not advocate general anesthesia in ocular surgery.
Ray K. Daily.

Rendahl, Ilmari. **The scotopic A-wave of the human electroretinogram. Clinical recording with the electronic flash as light stimulus.** Acta ophth. **36**:329-344, 1958.

Stimulation by electronic flash results in a diphasic electroretinogram even in dark-adapted eyes. A comparison between the b-potential of the standard ERG and

the negative a-potential of the electronic flash ERG shows the variations in these two potentials to be parallel. In the presence of circulatory disturbances of the retina, the a-wave is, however, considerably less affected. When the clinical ERG is supernormal, there seems to be an increase in both the a-and b-potentials on the electronic flash ERG. (10 figures, 40 references) John J. Stern.

Stanworth, A. **Modified major amblyoscope.** Brit. J. Ophth. **42**:270-287, May, 1958.

The author describes a modification of the major amblyoscope in which all the usual adjustments are present but in addition there is a system of mirrors and additional lenses which bring about an adjustment of the eyes which more nearly approximate the state of the eyes in normal usage. This makes possible more accurate measurements, particularly in detecting abnormal retinal correspondence and small-angle squints. Some details of examination and measurements on 17 patients are described. (7 figures, 17 references) Morris Kaplan.

Sundmark, Eric. **Recording of the human electroretinogram with the contact glass.** Acta ophth. **36**:273-280, 1958.

The electroretinogram was recorded with the use of a contact glass having nine electrodes placed along the same meridian. The b-potential was generally larger in the corneal part of the glass than in its scleral part. When a contact glass is used in recording the ERG, the recording electrode should be placed in its corneal part. (4 figures, 6 references) John J. Stern.

6

OCULAR MOTILITY

Esslen, E., Mertens, H.-G. and Papst, W. **The ocular myopathies.** Nervenartzt **29**:10-16, Jan. 20, 1958.

Little is known of the primary diseases of the ocular muscles and not much is found concerning them in German texts. The authors attempt to fill this gap with a series of articles, of which this is the first. The chronic progressive nuclear ophthalmoplegia of von Graefe is discussed here. The literature is summarized and a case is painstakingly analyzed from a clinical standpoint as well as by the newer techniques of electromyography and biopsy of several muscles. The authors find good evidence that von Graefe's ophthalmoplegia is a primary and widespread muscle disease rather than secondary to a nuclear disturbance. (4 figures 35 references) Edward U. Murphy.

Mackensen, G. **The speed of horizontal rotations of gaze.** Arch. f. Ophth. **160**:47-64, 1958.

Electrooculographic tracings of these movements show that the speed of movements is accelerated to just short of the half-way point where the speed reaches a maximum and then goes on to the new position of rest with equally gradual decrease of speed. Voluntary control of the rate of speed is not possible. The maxima of speed rise with the magnitude of deviation (14 figures, 4 tables, 21 references) F. H. Haessler.

Mertens, H.-G., Esslen E. and Papst, W. **Chronic ocular myositis.** Nervenartzt **29**:120-127, March 20, 1958.

This is the second in the series on ocular myopathies. Four cases are discussed in detail and the literature reviewed. Endocrine exophthalmos and orbital tumor are the two diseases this condition must be differentiated from and of great help here is an ocular muscle biopsy by "probe excision." The myositis usually responds well to cortisone therapy. (5 figures, 30 references) Edward U. Murphy.

Valvo, Giuseppe. **The importance of some systemic factors in the etiology of**

strabismus. Arch. di ottal. **61**:461-474, Nov.-Dec., 1957.

In a review of 205 clinic cases of strabismus the greatest emphasis was placed on infectious childhood diseases among etiologic factors. It was believed that preexisting heterophoria was converted to strabismus by the general malnutrition, diminished muscle tone, and reduced fusion sense during febrile illness. Of the cases reported, 186 were convergent and 19 were divergent. Eighty-five patients were male, and 120 were female. The author suggests that strabismus was ignored in males as cosmetically unimportant. While the history of age of onset depends on the observing power of the parents, there were extraordinarily many children with late onset. Sixty-four cases began in the first year, 41 in the second, 38 in the third, and 36 in the fourth. In perhaps 20 percent there was a family history of strabismus, and in another 20 percent of nervous or mental disease. (2 tables, 7 references) Paul W. Miles.

7

CONJUNCTIVA, CORNEA, SCLERA

Boberg-Ans, J. **Scleromalacia perforans.** Acta ophth. **36**:33-36, 1958.

The author describes a case of scleromalacia perforans in which one eye had to be enucleated and perforation and phthisis occurred in the other. It was unaffected by all treatment, including ACTH and cortisone. The etiology is completely unknown. (4 figures, 5 references) John J. Stern.

Charlin, C. **Recurrent keratoscleritis.** Arch. chil. de oftal. **39**:123-126, July-Dec., 1957.

The patient had recurrent episodes of scleritis associated with sclerosing keratitis and iridocyclitis with posterior synechiae. Most authors believe that the etiology is tuberculous, and antituberculous drugs are administered together with the local treatment of any keratitis or scleritis. The outlook in this disease is grave, as repeated episodes leave permanent damage in the eye. (4 references) Walter Mayer.

Forsius, Henrik. **Sensitivity of the cornea in arcus senilis.** Acta ophth. **36**:43-49, 1958.

In 18 subjects with intense arcus lipoides (juvenilis and senilis) in whom the fat had penetrated all layers of the corneal parenchyma, the corneal sensitivity was studied by means of a nylon thread (0.11 mm.); 19 without arcus, or if older with only microscopically visible or weak arcus, were used for comparison. The sensitivity is as a rule decreased in arcus. Sometimes there is a sector-shaped reduction as far as the region of the pupil in subjects of any age. (3 figures, 14 references) John J. Stern.

Heinmüller, G. **Fatty degeneration of the cornea.** Klin. Monatsbl. f. Augenh. **132**:566-568, 1958.

In a 49-year-old woman with a fatty degeneration of the right cornea the cholesterol level was high. (Two figures, 14 references) Frederick C. Blodi.

Oksala, A. and Lehtinen, A. **Diagnostics of rupture of the sclera by means of ultrasound.** Acta ophth. **36**:37-42. 1958.

Rupture of the sclera can be diagnosed and localized by means of ultrasonic waves, and the length of the rupture can be measured with an accuracy of 2 to 3 mm. The authors describe a clinical case and also experiments on bovine eyes. (5 figures, 8 references) John J. Stern.

Ruiz Barranco, F. **The treatment of pterygium.** Arch. Soc. oftal. hispano-am. **18**:234-240. March, 1958.

The literature on the etiology of pterygium is reviewed and it is emphasized

that the lesion is centered in the subconjunctival tissue, which is responsible for the recurrence and progression of the lesion. The author reports the results of treatment by repeated injections of hyaluronidase into the body of the pterygium in 60 cases. Satisfactory results were limited to cases of little activity. On the other hand Kamels operation, utilizing carbolic acid, was successful in 84 of 85 cases. The one case which recurred was cured by a repetition of the procedure. In large pterygia the author advocates a combination of Kamel's procedure with excision of the subconjunctival tissue. (1 table, 6 references)

Ray K. Daily.

Sbordone, G. and De Simone, S. **Clinical and pathogenetic considerations of keratoconus.** Arch. di ottal. **61**:497-504, Nov.-Dec., 1957.

Theories of the pathogenesis of keratoconus include the von Graefe glaucoma theory, endocrine or metabolic anomaly theory, mesenchymal dysgenesis, and avitaminosis. In this series the eye with keratoconus apparently had two to three millimeters higher intraocular pressure than the fellow eye in unilateral keratoconus. To ensure that the shape of the cornea did not affect tonometry, the Bailliart scleral tonometer was used in each of 20 patients with advanced keratoconus. It was repeated after caffeine and water provocation and after dilatation of the pupil. To rule out a hypersecretion type of glaucoma, each patient received fluorescein intravenously and the time of appearance in the anterior chamber was measured. Results showed no difference from the controls. Arterial retinal pressure also appeared normal, as did the blood pressure in these patients. The visual fields were all normal. The fundi showed no disc cupping, and only two showed myopic retinal changes.

In conclusion, keratoconus was attributed to mesenchymal dysgenesis, not glaucoma. (1 table, 7 references)

Paul W. Miles.

Straub, W., Sautter, H. and Velten, H. **Experimental and clinical findings after injury of the posterior surface of the cornea.** Arch. f. Ophth. **160**:26-42, 1958.

One to five parallel incisions of various length and depth were made into the posterior corneal surface of both eyes of each of 10 rabbits. The reactions of the cornea were observed with the slitlamp and are described in detail. Other than linear scars, the rabbit's cornea retains no permanent defects. On the other hand cruciform incisions leave extensive opacities, hence it seems advisable to avoid crossing incisions in the Sato operation for keratoconus. In a patient with astigmatism of lenticular origin the optically uncorrected vision was improved from 5/35 to 6/8 after two Sato operations. (21 figures, 1 table, 21 references) F. H. Haessler.

8

UVEA, SYMPATHETIC DISEASE, AQUEOUS

Fechner, P. U. **Spontaneous hyphaema with abnormal iris vessels.** Brit. J. Ophth. **42**:311-313, May, 1958.

In the eye of a woman who complained of sudden diminution of vision an engorged vessel was found near the sphincter of the iris with a microaneurysm from which a small, steady stream of blood flowed into the aqueous. The next day the bleeding had stopped and the anterior chamber was clear. No other abnormalities were found. (1 figure, 5 references)

Morris Kaplan.

Kessel, John F. **Uveitis caused by helminths and protozoa.** A.M.A. Arch. Ophth. **59**:854-860, June, 1958.

This is a brief review of the present literature on this subject and indicates

that such infestation does occur. (41 references) G. S. Tyner.

Lewallen, William M., Jr. **Aniridia and related iris defects.** A.M.A. Arch. Ophth. **59**:831-839, June, 1958.

In this report of 12 members in three generations of one family the author discusses the relationship to cataracts, glaucoma, and amblyopia. (4 figures, 1 table, and 21 references) G. S. Tyner.

Mayer, W. D. and Beitman, M. R. **Melanosarcoma of the choroid with a long period of survival.** A.M.A. Arch. Ophth. **59**:895-898, June, 1958.

The authors report the case of a patient who died of metastases 26 years after the removal of an eye for melanotic sarcoma of the choroid. (3 figures, 8 references) G. S. Tyner.

Zanen, J., Meunier, A. and Toussant, D. **Malignant melanoma of the ciliary body and the iris.** Bull. Soc. belge d'opht. **117**:538-553, 1957.

The many problems associated with malignant melanomas of the iris and the ciliary body are reviewed, especially the question of dissemination locally through the aqueous and by the bloodstream. In spite of the fact that early invasion of vessels is found in anatomic specimens, metastases do not have to occur. This variable and still unexplained local immunity had been apparent in several clinical cases when the carrier of malignant intraocular melanomas refused enucleation and nevertheless survived for many years.

A pigmented growth of the iris was found during a routine refraction in a 42-year-old woman. Gonioscopy revealed infiltration of the ciliary body, deformation of the lens and penetration into the chamber angle. The pathologic examination confirmed the clinical diagnosis; "malignant melanoma of the ciliary body with many spindle cells A and B and a few epithelioid cells." There were few reticular fibers, little pigment and no mitoses. The nuclei were, however, anaplastic and the outline of the cytoplasm poorly defined. The structure of the tumor was disorganized, especially where it approached the ora serrata. (13 figures, 21 references) Alice R. Deutsch.

9

GLAUCOMA AND OCULAR TENSION

Ancona, F. **Concomitant involvement of the ear and the eye in hypertensive vasculopathy.** Riv. oto-neuro-oftal. **32**: 678-686, Nov.-Dec., 1957.

The author evaluated the auditory system of 27 individuals with glaucoma; 20 of the patients had chronic glaucoma and seven had acute glaucoma. Hypertension of a high degree was present in each patient. A study of a similar group of hypertensive patients without glaucoma was also carried out. The studies did not substantiate the opinion expressed by others that there is a correlation between glaucoma and cochlear function. The author, however, emphasizes that his statistics are limited and that his negative results are not to be interpreted as an absolute refutation of the opinion of those who feel that there is a strong correlation between the eye and the ear in glaucoma. (9 references) William C. Caccamise.

Beretta, L. and Cerabolini, E. **The problem of hypacusia in glaucoma.** Riv. oto-neuro-oftal. **32**:662-277, Nov.-Dec., 1957.

The authors present a review of the literature together with an evaluation of their own patients in a study of the possible correlation between glaucoma and hypacusia. On the basis of their own clinical observations they conclude that a true pathogenetic relationship does exist. (3 figures, 24 references) William C. Caccamise.

de Carvalho, C. A., Lawrence, C. and Stone, H. H. **Acetazolamide (Diamox) therapy in chronic glaucoma.** A.M.A. Arch. Ophth. **39**:840-849, June, 1958.

This report from the Diamox Research Clinic of the Wilmer Institute covers the period from 1953 to 1957. Of 24 patients with chronic uncontrolled open-angle glaucoma, 21 were controlled by the addition of Diamox to the therapy. There is no evidence that long-term acetazolamide therapy irreversibly suppresses the formation of aqueous humor, for on discontinuance, the pressure rose again. It appears to be a relatively safe drug without severe side effects. The drug should not be used unless local agents have proved inadequate. This study deals solely with chronic simple glaucoma. Diamox is to be used in acute closed-angle glaucoma only as a preoperative procedure. (5 figures, 8 tables, 25 references) G. S. Tyner.

Espildora-Couso, J. **Tonography in a case of Marfan's syndrome.** Arch. chil. de oftal. **39**:118-120, July-Dec., 1957.

The author made tonographic measurements in a child with Marfan's syndrome and found that there was a definite abnormal resistance to the outflow of aqueous in the left eye and a questionable finding of resistance in the right. Repeated tensions taken with the Schiøtz tonometer were always normal. The author suggests that this resistance to outflow may be due to a trabecular lesion as a result of some mesodermic disturbance or to a poor function of the trabeculum produced by the subluxation of the lens.

Walter Mayer.

Ikuta, K. **Tonographic studies of human eyes. IV-VI.** Acta Soc. Ophth. Japan **62**:34-37, 149-152, 286-291, Jan., Feb., March, 1958.

In the first part, Ikuta describes tonography experiments which show that the aqueous outflow is inhibited by a contraction of the external ocular muscles. In the following parts he discusses the results of tonography in glaucoma. In glaucoma there is a definite delay in the aqueous outflow as shown by tonography. After filtration surgery the aqueous outflow looks normal for the first two minutes of tonography but the outflow becomes sluggish during the next two minutes. There occurs, therefore, a bending in the tonogram at the border of the first and the second half of the tonography. Ikuta considers that glaucoma can not be explained solely by a delayed outflow of the aqueous. (2 figures, 4 tables, 44 references) Yukihiko Mitsui.

Rivera Moreno, Antonio. **An investigation of differential tonometry as related to age and ocular pathology.** Arch. Soc. oftal. hispano-am. **18**:201-228, March, 1958.

By means of differential tonometry Marcello Carreras demonstrated the disparity between tonometric data and actual intraocular pressure. This procedure reveals the qualitative and quantitative tonometric error and permits the discovery of hypertension in some cases diagnosed as glaucoma without hypertension. The objective of this study was to determine what effect age and ocular diseases have on the differential tension. The procedure designated as differential tonometry comprises conventional tonometry, a tonometry performed while a pressure of 40 grams is applied to the globe with a Bailliart ophthalmodynamometer, calculation of the difference in the two data, and computation of the actual intraocular pressure. The author modified Carreras' original procedure by placing the angle between the tonometer and ophthalmodynamometer at 60 degrees instead of 45 as done by Carreras, and he set the pressure of the ophthalmodynamometer permanently at 40 grams. The material of the

investigations comprised 112 cases with 209 eyes. The data are tabulated and reported graphically according to age and acording to the ocular pathology of the examined eyes. The difference between the two tonometric measurements varied between 7 and 63 mm. Hg, which indicates that the same tonometric pressure may be found in two eyes with different actual intraocular pressures. The grouping according to age shows that the increase in the tonometric reading as a result of pressure on the globe diminishes with age; this indicates that for each tonometric reading the real intraocular pressure is higher, the older the individual. In children, on the contrary, high tonometric readings may correspond to a normal or low intraocular pressure. The intraocular pressure in many cases of simple glaucoma is actually not as high as is indicated by the tonometer. In corneal leucomas on the other hand the actual pressure is significantly higher than indicated tonometrically, and in such cases an elevated intraocular pressure can be overlooked. In spite of the indisputable value of differential tonometry as a research method and as a means of solving complicated clinical problems, the variations caused by age restrict its systematic clinical application. (17 figures, 23 tables, 8 references) Ray K. Daily.

Törnquist, R. and Broden, G. **Chamber depth in simple glaucoma.** Acta ophth. **36**:309-323, 1958.

The chamber depth was measured in 145 patients with simple glaucoma; the average was a little less than in normal eyes. This confirms earlier findings. The depth of the chamber in cases of long duration or after operation does not differ from others. It seems unlikely that the shallowness of the chamber is important in the etiology of simple glaucoma. (7 figures, 7 tables, 13 references)

John J. Stern.

Valcarce Avello, J. **Peripheral iridectomy in closed-angle glaucoma.** Arch. Soc. oftal. hispano-am. **18**:264-269, March, 1958.

The literature on the indications for peripheral iridectomy in closed-angle glaucoma is reviewed, and the results of this operation in 15 cases tabulated. It is concluded that the operation is indicated in early cases of acute and not very congestive closed-angle glaucoma, four to five days after the acute attack has subsided under medical therapy, and in the as yet normal fellow eye. (1 table, 2 references) Ray K. Daily.

10

CRYSTALLINE LENS

Atkinson, Walter S. **Selection of procedure for cataract extraction.** A.M.A. Arch. Ophth. **59**:811-817, June, 1958.

In this modification of his Academy lecture, Atkinson outlines his method and modifications in doing cataract surgery. He uses a limbus-based flap, replaced catgut sutures, uses his scleratome (similar to a cystatome) to make the nonpenetrating and the penetrating incision, enlarges with scissors, peripheral iridotomies and either forceps or the erisophake. In hypermature cataracts with glaucoma, not only should one attempt to get the tension down preoperatively, but should do the surgery very slowly to allow 20 minutes for decompression. (5 figures, 10 references) G. S. Tyner.

Dollfus, M. A., Haye, C. and Pinchon, S. **Complications and post-operative results of cataracts in diabetics.** Ann. d'ocul. **191**:209-219, March, 1958.

The authors analyze 94 cataract extractions performed in diabetic patients. Of these patients 41 percent obtained 5/10 vision or better, 21 percent from 1/10 to 5/10 and the remainder 1/10 or less. Complications were more frequent than in the

usual extraction; vitreous loss occurred in 8 percent, hyphemas in 16 percent and iridocyclitis in 12 percent. (1 table, 5 figures) David Shoch.

Eickoff, W. **Fibromatous pseudophakia.** Klin. Monatsbl. f. Augenh. **132**:526-532, 1958.

In this rare anomaly normal lens structure is replaced by fibrous or fatty tissue. One eye of a three-year-old boy remained small and blind. On enucleation loose vascularized fibrous tissue was found instead of the lens. No other major anomaly was present. (2 figures, 12 references)
Frederick C. Blodi.

Koskenoja, M. and Runeberg, C. **Does electric convulsive therapy cause cataract?** Acta ophth. **36**:102-109, 1958.

Of the 633 patients in a mental hospital whose eyes were examined, 237 had received electric convulsive therapy. The statistical analysis of the findings did not demonstrate that this therapy produces cataract. (4 tables, 32 references)
John J. Stern.

Lieb, W. A. and Guerry, DuPont, III. **Anterior chamber lenses.** Klin. Monatsbl. f. Augenh. **132**:465-475, 1958.

The authors discuss the use of these lenses in unilateral aphakia. Animal experiments and 16 patients are reported. The plastic lens has two nylon loops for support. (15 figures, 13 references)
Frederick C. Blodi.

Roeser, Johannes. **Comparison of operations with and without the lytic cocktail.** Klin. Monatsbl. f. Augenh. **132**:475-480, 1958.

The authors operated upon 220 patients in each group. The sedative effect of this drug mixture was much greater than the standard method. The eye became softer, but the percentage of intracapsular extractions was reduced from 85 percent to 75 percent. A retrobulbar injection became superfluous. The incidence of complications decreased, for example the loss of vitreous from 13.6 percent to 9.1. (5 tables, 12 references)
Frederick C. Blodi.

Sbordone, G. **Caloric cataracts, a clinical study.** Arch. di ottal. **61**:487-496, Nov.-Dec., 1957.

One case is reported which occurred in a man aged 47 years. The diagnosis was based on the biomicroscopic appearance of the lenses, and not necessarily on a history of exposure to infrared radiation. This is important, because a cataract may be delayed as much as three years after industrial exposure has terminated. The pathognomonic changes are: 1. superficial breaks in the lens capsule, 2. subcapsular vacuoles and anterior cortical radial striae, and 3. posterior subcapsular opacities, often polychromatic. As the cataract matures, the nuclear changes are no different from those of senile cataract.

Whether caloric cataracts are due to direct action on the lens is debatable. The most likely clue is the fact that the opacities are in the zones back of iris pigment, and not in the exposed pupil area. (6 refences) Paul W. Miles.

Sgrosso, Salvatore. **Operations and operators of cataracts. Variations of the Graefe incision.** Arch. di ottal. **61**:437-444, Nov.-Dec., 1957.

In this essay on physician-patient relations, preoperative care, operative technique, and postoperative complications of cataract, the author discusses the timid operator who uses eye drops and postpones the operation and gives advice as to how to handle all the patient's relatives and friends who want to take part in the event. He recommends that the surgeon, as he enters the operating room shake hands with all the assistants and aids and smile, to be sure of the best spirit while operating. Paul W. Miles.

11
RETINA AND VITREOUS

Beyer, Eva-Maria. **Familial tortuosity of the small macular arteries with hemorrhages.** Klin. Monatsbl. f. Augenh. **132**: 532-539, 1958.

This anomaly was observed in a 47-year-old man and his 17-year-old son. Repeated macular and paramacular hemorrhages occurred. No systemic affection was found. (4 figures, 11 references)

Frederick C. Blodi.

De Jaeger, A. and Bernolet. **Sclerectomy in retinal detachment.** Bull. Soc. belge d'opht. **117**:554-560, 1957.

After a short survey on the various techniques in scleral shortening operations the authors describe their own modified procedures of scleral resection. Routine pre-surgical preparations, conjunctival incision and dissection of Tenon's capsule, and looping of the corresponding external muscles is followed by a partial scleral resection in the region of the retinal holes. The resected part of the sclera should be 3 to 4 mm. broad and embrace about one-half of the circumference of the eye. It is stressed that the scleral incisions should be slanting. This type of scleral cleavage makes the closure of the wound easier, as the superficial layers of the sclera contain more elastic fibers than the deep layers and therefore can be approached under less tension. A very light electro-coagulation should be performed over the resected part. The scleral wound is closed with 5-0 plain catgut. Immediately after surgery the retina is often found to have remained detached. In many cases the retina was reattached only after several months. (16 references)

Alice R. Deutsch.

Dodo, T. and Toda, S. **Vitreous replacement as a treatment of severe vitreous opacity.** Acta Soc. Ophth. Japan **62**:129-143, Feb., 1958.

The vitreous with severe opacities was replaced by clear vitreous or by spinal fluid in 13 human eyes. In most cases a definite clearing and improvement in the vision was obtained. A replacement of as large an amount as two ml. was most effective. A vitreous aspiration alone did not show such a good effect. The replacement seemed to facilitate further absorption of the opacity. Side-effects were not common. Hemorrhage into the vitreous and iritis occurred in a small number of cases. (1 table, 17 references)

Yukihiko Mitsui.

François, J. and De Rouck, A. **Electroretinography in the differential diagnosis of tapeto-retinal degeneration.** Bull. Soc. belge d'opht. **117**:511-538, 1957.

Bio-electric disturbances, which are recorded in retino-encephalography, were found in diffuse and localized tapeto-retinal degenerations. They included abnormal reactions of the retina and the cerebral cortex in varying degrees, and associated with each other or as separate particular deviations. In pigmentary retinopathy, in retinopathia punctata albescens and in choroideremia the bioelectric syndrome was complete. This means the retina did not react or reacted minimally to any light stimulation, the conduction and reception in the cortex was retarded and the EEG was diffusely disturbed. Degeneration of the central retina caused modifications in the ERG of various degrees. The ERG was rarely abolished but often considerably impaired. It was practically absent in several cases of central pigmentary retinopathy and subnormal in Sorsby's dystrophy. In angioid streaks it was found both normal and subnormal and the same was true for senile degenerations of the macula. The EEG was normal in the latter. The electro-retinography and encephalography proved to be essential for the differential diagnosis between the different groups of degenerative retinal dis-

eases. The presence or absence of the ERG is also important for the prognosis and it was considered to be more significant than visible changes in the funds. (17 figures, 59 references)

Alice R. Deutsch.

Jampel, R. S. and Falls, H. F. **Atypical retinitis pigmentosa, acanthrocytosis, and heredodegenerative neuromuscular disease.** A.M.A. Arch. Ophth. **59**:818-820, June, 1958.

Only three cases of this disease have been reported. This paper gives a discussion and follow-up information on these patients. (2 figures, 4 references)

G. S. Tyner.

Kornerup, Tore. **Retinopathia diabetica proliferans.** Acta ophth. **36**:97-101, 1958.

Among the 1,402 unselected diabetic patients, retinopathia diabetica proliferans was seen in 117 or in 8.4 percent. These patients were compared with other diabetics. There was no significant difference in age and duration of the diabetes. In 78 percent of the patients with the retinopathy, ophthalmoscopically discernible venous changes were found; in this respect they differ significantly from other diabetics. Heredity is not a more important factor in these cases. The majority of patients with the retinopathy have an onset of diabetes in childhood and the relative systolic and diastolic blood pressure is definitely higher.

Proteinuria is twice as frequent as in other diabetics; fundus hypertonicus is difficult to diagnose in patients with this retinopathy but there is no reason to assume that it is more frequent. Insulin treatment does not differ from that given to other diabetics. This retinopathy is not a further stage of development of other types of retinopathy; cases are reported in which it was the first sign of retinal change. Its presence in pregnant women does not invariably lead to stillbirth or spontaneous abortion. Of the patients with this retinopathy 28 percent were blind.

Proliferative retinopathy is more frequent among patients on a full diet than among those on a restricted diet. The effect of hypophysectomy and adrenalectomy are briefly mentioned and the latter does not seem to arrest the progress of the proliferative retinopathy. (4 tables, 63 references)

John J. Stern.

Vanysek, Jean. **The ERG in circulatory disturbances of the retina.** Bull. mém. Soc. franç. d'opht. **70**:322-345, May, 1957.

One of the main purposes of clinical retinography is the establishment of a relationship in between circulatory disturbances of the retina and correlative activity of the retina. A characteristic curve of four phases (P-1, P-2, P-3, P-4) was seen in the ERG during the application of the ophthalmodynamometer and related to the venous retinal pressure, retinal dyastolic and retinal systolic pressure, respectively. In normal subjects and in hypertensives without fundus lesions the general aspect of these graphs was identical. Pathologic signs were outlined after an analysis of 100 examinations which included cases of essential hypertension, arteriosclerosis, embolism of the central retinal artery, thrombosis of the internal carotid and thrombosis of the retinal veins. In congestive glaucoma and in the initial stage of essential hypertension the ERG is supernormal, an analogy to the ascending phase P-1. The supernormal b-wave in arteriosclerosis of the retinal vessels and in advanced stages of essential hypertension present an analogy to the phases P-2 and P-3. The extinction of the ERG after abolition of the systolic tension in the retina is similar to the extinction of the ERG in an embolism of the main stem of the central retinal artery or in thrombosis of the internal carotid. The reëstablishment of a normal ERG after

abolition of the retinal circulation with the dynamometer occurs much sooner in hypertensive patients than in normal subjects. By itself the ERG cannot explain problems of circulation but in combination with other types of examination it is an essential guide towards a better understanding of circulatory disturbances in the retina and of allied clinical pathologic situations. (15 figures, 19 references)

Alice R. Deutsch.

Vena, Antonio. **Treatment of retinal thrombophlebitis with vasodilators and anticoagulants.** Arch. Soc. oftal. hispano-am. **18**:270-274, March, 1958.

The literature on the subject is reviewed and two cases reported of thrombosis of the central retinal vein, of longer than 20 days duration, successfully treated by a combination of anticoagulants and vasodilators. In one case vision improved in the course of two months from 1/10 to 1/2. In the other case the retinal edema increased under therapy with anticoagulants alone; after the addition of vasodilators to the therapeutic regimen the condition began to improve immediately and after two months vision improved from the initial 1/20 to 1/10. Heparin was the anticoagulant used, and for vasodilation intravenous injections of novocaine were used in one case, and X-ray irradiation according to Desjardins' technique in the other. (15 references)

Ray K. Daily.

12

OPTIC NERVE AND CHIASM

Bayet, T., Brihaye, M. and Van Geertruyden. **Retrobulbar injections of hydrocortisone in optic neuritis and retrobulbar neuritis.** Bull. Soc. belge d'opht. **117**:487-506, 1957.

Retrobulbar hydrocortisone (12.5 mg.) was used in four patients with retrobulbar neuritis and optic neuritis respectively. In three patients the disease was ascribed to inflammatory processes; one patient who had several episodes of this disease and also had paresthesias of the lower extremities was thought to have multiple sclerosis. The local treatment compared favorably with general. Improvement could be noticed immediately and recovery was achieved in 7 to 14 days. This beneficial effect, however, occurs only when treatment is given without delay. Whenever the start of the local treatment was delayed the good results were much slower. A detailed general and laboratory work-up was done in all four cases. Systemic treatment consisted of penicillin and streptomycin. Retrobulbar hydrocortisone was the only local treatment. Early retrobulbar injection of hydrocortisone is the treatment of choice in both optic neuritis and retrobulbar neuritis. (10 figures, 34 references)

Alice R. Deutsch.

Iverson, Herman A. **Hereditary optic atrophy.** A.M.A. Arch. Ophth. **59**:850-853, June, 1958.

The author reports five definite cases of hereditary optic atrophy in four generations of a family. The heredity is dominant, and the condition appears to be congenital. (2 figures, 6 references)

G. S. Tyner.

Lasco, F. **Pseudo-glaucoma following methyl alcohol intoxication.** Ann. d'ocul. **191**:235-241, March, 1958.

Two cases of methyl alcohol poisoning are reported which resulted in a typical glaucomatous atrophy of the optic nerve heads. Repeated measurement of tension and provocative tests ruled out glaucoma. No calcification of the carotids could be seen radiologically. The vision improved eventually in spite of the appearance of the discs, which indicates that some fibers had escaped permanent damage.

David Shoch.

13

NEURO-OPHTHALMOLOGY

Amidei, B. and Megighian, D. **Electronystagmographic analyses of congenital nystagmus.** Riv. oto-neuro-oftal. **32**: 687-700, Nov.-Dec., 1957.

The authors report their electronystagmographic findings in congenital horizontal nystagmus. Emphasis is placed on the changes in the E.N.G. resulting from various head positions. (3 tables, 21 references) William C. Caccamise.

Amidei, B. **The possible inhibition of central congenital nystagmus.** Riv. oto-neuro-oftal. **32**:701-708, Nov.- Dec., 1957.

The author observed some patients with congenital nystagmus which was inhibited when they were writing. He attributes this to an effect of the attention factor on the bulbo-mesencephalic system. (2 tables, 10 references)
William C. Caccamine.

Bynke, Hans. **The syndrome of Foster Kennedy.** Acta ophth. **36**:129-140, 1958.

Foster Kennedy's syndrome (optic atrophy with central scotoma of one eye, papilledema of the other) was found in 16 out of 1,600 patients with cerebral disease. The syndrome occurred with brain tumors as well as brain abscess or optic neuritis. The infrequence and restricted diagnostic value of the syndrome are emphasized. (4 figures, 1 table, 38 references) John J. Stern.

Espildora-Couso, J. **Paracentral homonymous hemianopic scotoma.** Arch. chil. de oftal. **39**:121-122, July-Dec., 1957.

A patient complained of difficulty in vision during an episode of endocarditis. Examination was negative with the exception of a left homonymous hemianopsia. Two months after the first visit the patient again complained that he had difficulty in reading; he seemed to be unable to find the line below the one he was reading. At that time, examination revealed a perfectly normal peripheral field, without any sign of hemianopsia and when tested with the Bjerrum screen a paracentral left inferior hemianopic scotoma was found

The author ascribes these findings to an embolus in the posterior calcarine artery, which completely inhibited the right occipital cortex. The complete hemianopsia is explained by a temporary cortical inhibition due to reflex vascular spasm. (1 figure) Walter Meyer.

Junceda Avello, J. **A case of Leber's disease.** Ann. d'ocul. **191**:228-234, March, 1958.

The author concludes, on the basis of this case which he reports and a review of the literature, that Leber's disease is a hereditary lesion of the central nervous system in which other manifestations may be found. He doubts that infection is a factor and feels that reported cases of chiasmal arachnoiditis may be secondary to the optic atrophy. He also suggests that there may be a hereditary tendency to chiasmal arachnoiditis in some cases. (3 figures) David Shoch.

Troupp, H., Koskinen, K. and af Björkesten, G. **Ophthalmoplegia caused by intracranial aneurysm.** Acta ophth. **36**:79-86, 1958.

A series of six patients with aneurysm of the internal carotid artery causing oculomotor palsy is reported. In addition, two instances of aneurysms in other locations causing oculomotor palsy are reported. All of these patients were subjected to intracranial operation with ligature or clipping of the neck of the aneurysm. The shortest follow-up was one year. Not one patient recovered completely. It seems that the only chance to improve the poor prognosis is to operate in the acute stage of the disease. (1 figure, 18 references)
John J. Stern.

Watillon and Pivont, A. **The value of contact lenses for the treatment of a paralytic ptosis associated with a facial paralysis.** Bull. Soc. belge d'opht. **117**:608-614, 1957.

External progressive ophthalmoplegia is a hereditary progressive disease, which resembles congenital hereditary ophthalmoplegia and the infantile form of progressive bulbar paralysis. The first symptoms occur in childhood or adolescence and only rarely after the age of forty. Only the external eye muscles are affected. The patient discussed was 45 years old. A progressive ptosis and impairment of elevation and abduction started in 1955 and was followed by a bilateral facial paralysis. The coexistence of ptosis and bilateral facial paralysis was a difficult therapeutic problem. Specially constructed plastic contact lenses with a rim above to support the upper lids enough to free the pupillary area were used. The whole lens rested in the inferior fornix which supported both the lens and the upper lid. The lenses have been worn for six to eight hours a day for more than a year with only minor discomfort. The functional result and the external appearance were excellent. (3 figures, 15 references) Alice R. Deutsch.

14

EYEBALL, ORBIT, SINUSES

Dorello, U. **The eye and the sinuses.** Riv. oto-neuro-oftal. **32**:613-633, Nov.-Dec,. 1957.

The author discusses the relationship between diseases of the sinus and of the lacrimal ducts. A systematic X-ray study of 500 cases of lacrimal duct disease revealed sinus changes in 107 (21.4 percent). From his study the author feels that sinus conditions should be considered as a possible factor in those patients who have stenosis of the nasolacrimal duct. It is suggested that treatment of the sinus condition may prevent further progress of the disease in the lacrimal duct. (10 figures, 2 tables, 32 references) William C. Caccamise.

Erdmann, H. and Erdmann, R. **Orbital complications with sinus affections in infants.** Klin. Monatsbl. f. Augenh. **132**:559-566, 1958.

A seven-weeks-old infant developed an orbital abscess secondary to a purulent infection of the ethmoids and the antrum. The orbit had to be opened. (7 figures, 5 tables, 19 references) Frederick C. Blodi.

Goodman, E. and Pond, M. J. **Serous tenonitis.** A.M.A. Arch. Ophth. **59**:899-900, June, 1958.

Serous tenonitis in a 13-year-old school girl was treated successfully by the administration of Prednisolone and Gantrisin by mouth and Terracortril ophthalmic suspension as a collyrium. The authors suggest that the orbital synovia may be selectively attacked by rheumatic conditions, just as joints and tendons are. (1 reference) G. S. Tyner.

Pasmanik, S. **Orbital lymphosarcoma.** Arch. chil. de oftal. **39**:110-112, July-Dec., 1957.

A patient came to the clinic with a tumor over the left lacrimal sac, which had been present for five months. Biopsy revealed a neoformation of lymphatic tissue, which could have been leukemia or lymphosarcoma. The first disease was ruled out by the hematologist and the patient received a total of 9000 r in 40 sessions over 62 days. Eight months later the mass had totally disappeared and there was no evidence of any neoplasm.

The author briefly discusses the pathology of lymphatic tumors. He feels that radiotherapy is the treatment of choice, but even with it, there are often late metastases. Even in the case where sur-

gery is used as a primary procedure, radiotherapy must be given after it, no matter how extensive the primary surgery was. (4 references) Walter Mayer.

Peters, G. and Seitz, R. **Endophthalmitis in congenital toxoplasmosis.** Klin. Monatsbl. f. Augenh. **132**:540-548, 1958.

One eye was enucleated in a six-weeks-old child with congenital toxoplasmosis. The clinical picture somewhat resembled a retinoblastoma. An endophthalmitis with complete retinal detachment was found in this microphthalmic eye. Organisms were seen in the retina and in the choroid but no culture had been taken. The child died at the age of one year. (5 figures, 25 references)

Frederick C. Blodi.

Pietruschka, G. **Further comments on the marble bone disease.** Klin. Monatsbl. f. Augenh. **132**:509-525, 1958.

This is an addition to an earlier paper on the same subject (Klin. Monatsbl. f. Augenh. 123:189). A fourth case is described. The 22-year-old girl also had optic atrophy, exotropia and exophthalmus. One of the three previously described patients died in the meantime. In the differential diagnosis fibrous dysplasia, mandibulo-facial dysostosis and hyperostosis have to be considered. (15 figures, 23 references)

Frederick C. Blodi.

Sebestyen, J. **Therapeutic results in acute orbital cellulitis.** Klin. Monatsbl. f. Augenh. **132**:480-486, 1958.

This is a comparison of 25 cases treated before the advent of antibiotics and sulfonamides and 25 cases treated after it. The mortality rate in the early period was about 18 percent; it sank to 12 percent with sulfonamides, and with antibiotics to 4 percent. Complete recovery occurred in the early period in 36 percent of the treated cases and later in 68 percent. (1 table, 50 references)

Frederick C. Blodi.

Valenzuela, R. **Exophthalmos due to chronic orbital granuloma.** Arch. chil. de oftal. **39**:127-134, July-Dec., 1957.

The clinical history of two patients with exophthalmos, pain in the affected eye, decrease in visual acuity, and negative X-ray findings is described. In both cases a biopsy was done and chronic inflammatory tissue was found. Both patients were treated with radiotherapy with good results; as the exophthalmos regressed the visual acuity improved. In the first case a time interval of more than eight years elapsed between the time a biopsy was advised and the patient agreed to have it done, and in spite of this long interval, the recovery in acuity was good.

The author feels that radical surgery is contraindicated in such cases and that radiotherapy does not act directly on the fibrotic tissue, but by inhibiting the vascular elements of the granuloma and destroying the lymphatic tissue of the tumor. (7 figures, 3 references)

Walter Mayer.

15

EYELIDS, LACRIMAL APPARATUS

Misar, Rainer. **The treatment of dacryostenosis with cortisone fillings.** Klin. Monatsbl. f. Augenh. **132**:498-504, 1958.

In patients with narrowing of the lacrimal duct dilation is first done under general anesthesia. Then a mixture of cortisone, Irgamid and terramycin ointment is put into the duct. In this manner 29 patients were treated with success. Usually three fillings are necessary to keep the duct open, but occasionally as many as 12 had to be given. Recurrences in eight patients were treated successfully.

Frederick C. Blodi.

Nagashima, K. **Studies on the function of the lacrimal pathway.** Acta Soc. Ophth. Japan **62**:9-19, 578-596, Jan., May, 1958.

By means of a special manometer designed for this study the author measured the pressure in the lacrimal sac and lacrimal ducts. He demonstrates that the sac and ducts show a pumping action and the action is induced by lid movements. In general a positive pressure occurs with lid closure and the converse with lid opening. The whole lacrimal pathway shows peristaltic movement. By one movement on the average, 0.2 cmm. of tears is carried. The pumping action of the lacrimal ducts is preserved in the early stage of lacrimal pathway stenosis and obliteration but disappears in the late stage. Application of a vinyl-tube as a treatment of stenosis of the lacrimal pathway is effective in the early stage of the condition, where the pumping action is preserved. The pumping action disappears when the facial nerve is paralyzed. (2 figures, 10 tables, 81 references)

Yukihiko Mutsui.

Pujol Canicio, Alberto. **Acute bilateral tuberculous dacryoadenitis.** Arch. Soc. oftal. hispano-am. **18**:241-254, March, 1958.

The author reports a case of acute bilateral dacryoadenitis in a woman 35 years old. In addition to general symptoms of an acute infection and a stomatitis, she had a bilateral painful swelling of the upper lids in the region of the lacrimal glands, intense bilateral conjunctival bulbar and palpebral congestion, and chemosis. The left lateral rectus muscle was paretic and the patient had a paralytic convergence. The refractive media were somewhat opaque; this is attributed to pressure on the globe. The diagnostic examinations revealed no specific information. The patient recovered under treatment with streptomycin and hydrazide. The differential diagnosis is discussed and it is concluded that this was a rare case of acute tuberculosis of the lacrimal glands. (7 references)

Ray K. Daily.

Sédan, Jean. **Classification of lacrimal duct potency.** Ann. d'ocul. **191**:220-227, March, 1958.

The author divides cases of epiphora into four groups according to the status of the naso-lacrimal duct: spontaneously patent, provoked patency, forced patency, and obstructed. He analyzed 1,939 cases and found about as many in obstructed ducts as he did ducts with some degree of patency. Of the 973 patients with obstruction, 849 were cured without surgery. The author emphasizes the need for repeated probing, irrigation, the use of cortisone and antibiotics locally, and, most important, treatment of the nasal passages.

David Shoch.

16

TUMORS

Appelmans, M., Michiels, M., Dehoux, M. and Van Hoonacker, E. **Ocular manifestations of the angiosarcoma of Kaposi.** Bull. Soc. belge d'opht. **117**:617-630, 1957.

Multiple purple cutaneous nodules, sharply outlined but nevertheless frequently of rapid growth, are the main clinical characteristics of Kaposi's sarcoma. The nodules may be confluent and form larger tumors but the skin mostly remains movable with the tumors although it often gets thin and atrophic in these areas. Telangiectases are frequent and ulcerations rare. The inferior extremities are almost always the primary seat and symmetrically affected. Lesions on the face and the mucous membranes are rare and considered to be an ominous foreboding. Pathologically the disease is a malignancy of the vascular system, of

multiple origin but not inclined to metastasize or to be transplanted into the regional lymph glands. The lesions are polymorphous and vary not only in different individuals but even in the same individual. This histologic picture is characterized by fusiform elements often in rows and surrounded by reticulin-fibers with pericytes, histiocytes and plasmacytes in various numbers and state of mitosis among numerous new-formed capillaries and blood filled sinuses. Localized proliferation of the endothelium, followed by thrombosis, explain the occasional fibrosis of the nodules. The course is mostly slow and death often occurs as a result of intercurrent disease. Kaposi's sarcoma is rare in Europe. It is frequently seen in central Africa. Men of middle age are most often affected.

The history of a 79-year-old man is reported. His main complaint was a tumefaction of the left upper lid. Similar tumor nodules were found on his lower extremities. The diagnosis was confirmed by biopsy of a skin nodule. The lesions of the upper lid showed areas of necrosis and ulceration, rare findings in angiosarcoma. Chemotherapy, hormones, penicillin and radiotherapy were of no benefit as more and more nodules appeared. The patient died of suffocation after six months. (6 figures, 21 references)

Alice R. Deutsch.

Kurus, Ernst. **A contribution to the knowledge of the giant-follicular lymphoblastoma.** Klin. Monatsbl. f. Augenh. **132**: 568-571, 1958.

The author describes a 78-year-old woman with an orbital lymphoma of the Brill-Symmers type. After the tumor was excised irridation was given. (4 figures, 8 references) Frederick C. Blodi.

Marsico, Vincenzo. **Clinical and histologic contributions to the study of "mixed tumor" of the orbital region.** Arch. di ottal. **61**:445-454, Nov.-Dec., 1957.

Recent reports about "mixed tumor" of the orbit have emphasized characteristics of both lacrymal and salivary glands; 25 percent were said to be potentially malignant. The case of a 52-year-old woman is reported. She had a walnut-sized tumor in the area of the lacrymal gland. Clinically it was found to be two-thirds outside the orbital rim, smooth, hard, elastic, movable, and painless; histological examination revealed a mixed tumor of the salivary type. It was considered to be a growth from embryonic gland tissue or cell rests, and not metastatic. (2 figures, 20 references)

Paul W. Miles.

Otto, Joachim. **Basal cell epithelioma in patients wearing glasses.** Klin. Montasbl. f. Augenh. **132**:504-508, 1958.

Four patients are reported who developed an epithelioma in an area which was constantly irritated by the spectacle frame. This points toward a close connection between trauma and tumor. (4 figures, 3 references) Frederick C. Blodi.

Sundmark, Eric. **The electroretinogram and malignant intraocular tumors.** Acta ophth. **36**:57-64, 1958.

Electroretinography seems to be of no value for the differential diagnosis of intraocular tumors. (3 figures, 1 table, 5 references) John J. Stern.

17

INJURIES

Law, Frank W. **Insect sting of the lid and a sequel.** Brit. J. Ophth. **42**:314-315, May, 1958.

A six-year-old child was stung on the lid by a bee and when examined two days later no evidence of the sting or of corneal damage could be found. Four days later much general irritation and horizontal

scratches of the cornea were present. The stinger of a bee was found protruding through the tarsus and rubbing on the cornea. It was easily removed. The interesting observation is made that the horizontal movements of the eye resulted in much more scratching than the vertical ones. (2 figures, 1 reference)

Morris Kaplan.

Levy, Walter J. **Peforating ocular injury: a long-term follow-up.** South African M. J. **32**:293-295, March 15, 1958.

In a decade 1,303 cases of perforating ocular injury, not due to intraocular foreign body, were admitted to Moorfields Eye Hospital, and of these, 511 cases have been followed for a minimum period of three years and an average of five and one-half years and are here reported. A low percentage of 11.7 percent (153 cases) of 1,303 cases lost an eye. Forty percent achieved 6/16 or better vision, and the prognostic effect of damage to the lens is stressed in relation to visual results and to retention of the eye; the damage causes a drop in the percentage of those with a 6/16+ result from 70 percent in the group with clear lens to 12.6 percent in the cataractous group. The stability of localized lens opacity is remarked upon.

The tragically high number of children who suffer perforating ocular injury, often with loss of an eye, particularly as a result of accidents with scissors, knives, darts or arrows (almost all preventable) is emphasized.

The surgical technique usually used in Moorfields is detailed and the use of sterile air or saline for anterior-chamber re-formation is advocated. (8 tables, 3 references) Author's summary.

18

SYSTEMIC DISEASE AND PARASITES

Bodenhäuser, Joe Henry. **Uncommon findings in temporal arteritis and an etiologic suggestion.** Arch. f. Ophth. **160**:113-124, 1958.

A woman, 78 years of age, had typical manifestations of temporal arteritis with blindness of one eye. At the height of the illness numerous red spots appeared in the skin in the area supplied by the temporal and maxillary arteries. Histologic study showed perivascular infiltration of the precapillaries and extensive capillary ectasia on the basis of which the author offers a theory which clarifies the various manifestations of temporal arteritis. (3 figures, 46 references) F. H. Haessler.

Fornes Peris, E. **Therapy of Sjøgren's syndrome on an etiologic basis.** Arch. Soc. oftal. hispano-am. **18**:229-233, March, 1958.

A case of Sjøgren's syndrome is reported, in which definite improvement was obtained by increased sexual activity and the administration of Perandren. The literature on the pathogenesis and treatment of this syndrome is reviewed, and it is concluded that the disease is caused by a hormonal insufficiency.

Ray K. Daily.

Gregersen, E. **Rubella embryopathy involving dehiscence and mesodermal aplasia of the iris.** Acta ophth. **36**:110-114, 1958.

The author reports a case of rubella embryopathy in the form of mesodermal aplasia and dehiscence of the iris associated with embryopathic pigmentary degeneration of the retina and patent ductus arteriosus. Rubella embryopathies as well as the origin and clinical features of the present malformations are briefly discussed. (1 figure, 22 references)

John J. Stern.

Jensen, V. J. **Dermo-chondro-corneal dystrophy.** Acta. ophth. **36**:71-78, 1958.

The following signs were observed in a seven-year-old boy, 1. bilateral, periph-

eral, subepithelial corneal opacities of a dystrophic nature and unilateral pterygium. 2. multiple, fairly small, firm, yellowish skin tumors, and 3. osteochondral changes in the peripheral bones of the extremities. In spite of some differences, the syndrome is in many respects similar to that of familial dermochondrocorneal dystrophy described by François and probably is a manifestation of the same nosologic entity. (7 figures, 4 references)

John J. Stern.

Kessel, John F. **Observation on the methylene blue dye test for toxoplasma.** A.M.A. Arch. Ophth. 59:861-867, June, 1958.

After a review of the literature and his experimental work, the author believes that the methylene blue dye test for T. gondii is specific. A comparison of dye-test results with toxoplasmin skin tests results shows a high correlation between tests which are dye-test negative. (5 tables, 31 references) G. S. Tyner.

Lobstein, André. **Dynamic aspects in retinal and cerebral circulation.** Bull. mém. Soc. franç. d'opht. 70:308-321, May, 1957.

Kety's test is one of the procedures which can provide quantitative data on the relationship between circulatory factors and functional activity of the retina. It is based on the principle of Fick which concerns differences of concentration of an inert gas in cerebral arteries and in cerebral veins after having been fixed by the brain during a given time. This difference is inversely proportionate to the amount of blood moving across the brain. The course of the investigation is described in detail and the possible modifications of intracranial pressure, changes in blood pressure and in caliber of cerebral arteries together with the nervous, humoral and organic forces regulating them, are discussed. The various hemodynamic formulae and their interrelationship are explained and the inevitable difficulties and complications in this type of research are recognized. Three essential factors definitely in direct correlation are elevation of the blood pressure, fundus abnormalities and the level of cerebral-vascular resistance; but even this parallelism is not absolute. The combination of these different types of examination, objective establishment of the cerebral blood-flow, dynomometry and functional quantitative perimetary tests may be of practical clinical value in the choice of treatment in given cases. (1 figure, 1 table, 76 references) Alice R. Deutsch.

Matsui, M. **The relation between retinal blood pressure and retinal function in hypertensive patients.** Acta Soc. Ophth. Japan 62:28-34, 257-262, 316-322, 726-733, Jan., March, June, 1958.

This is a study of the relationship between the retinal blood pressure and the renal function. In any type of hypertension there is a negative correlation between the renal blood flow and the clinical rigidity of the retinal arteries. A correlation between the renal blood flow and the retinal blood pressure is present in renal hypertension but not in essential hypertension. A depression of blood pressure by such medicaments as Reserpin and 1-hydrazinophthalazine, causes a lowering of retinal blood pressure with an increase in renal blood flow. An excessive administration of the medicaments may cause a decrease in renal blood flow.

Matsui also describes two cases of hypertensive retinopathy in which renal function was not disturbed. He considers that a disturbance in renal function is not essential for the manifestation of hypertensive retinopathy. (26 figures, 9 tables, 46 references) Yukihiko Mitsui.

Palm, Erik. **The ocular crisis of the temporal arteritis syndrome. (Horton).** Acta ophth. 36:208-243, 1958.

Palm presents 31 cases of temporal ar-

teritis with severe ocular complications from various Swedish hospitals. In 13 cases the diagnosis was based on biopsy, in 13 others on typical clinical manifestations and in five cases the diagnosis was highly probable but not certain. Among the 26 cases with a certain diagnosis, only eight had typical temporal changes while in 12 these were uncertain and in six absent. Thirty patients had severe impairment of vision (finger counting or less), in nine cases in one eye, and in 21 in both. In one case the functional disorder was limited to a field defect. The majority had ischemic papilledema; four eyes had embolism of the retinal vessels, and in six eyes both embolism and papilledma were observed. In one case the ischemic process was pruely retro-bulbar in both eyes and the fundi were normal until atrophy set in. In two cases the site of the ischemic lesion was verified electroretinographically. In two cases a strong systolic bruit was heard over the cranium. In one of these, there was marked bilateral stenosis of the internal corotid artery, 1 cm. long, at the origin of the ophthalmic artery. The author stresses the frequency with which the disease appears in a form almost lacking in typical symptoms; the disease takes an ocular character. Ischemic papilledema in association with a sudden loss of vision and a high sedimentation rate in an elderly patient should be considered almost pathognomonic for this disease. The treatment seems to be powerless against established opticoretinal ischemia. ACTH or cortisone, however, have been proved to be of great prophylactic value. The second eye was injured in only one of eight patients adequately treated with cortisone whereas in 17 not treated the second eye was affected in 15. Every patient with temporal arteritis, whether he has ocular symptoms or not, should receive prophylactically ACTH or cortisone as long as the sedimentation rate is high. (2 figures, 6 tables, 55 references) John J. Stern.

Schulze, E. **Summary: Behçet's disease.** Deutsche med. Wchnschr. **83**:469-470, March 21, 1958.

Fifteen years before Behçet's report in 1937, iridocyclitis had been described in association with mucous membrane lesions. Recent research into this condition led to its establishment as a virus disease. The pathology of the complicating encephalomyelopathy is described. The brain lesions are vascular and apparently thrombotic. They do not resemble the changes seen in the demyelinating diseases. (12 references) Edward U. Murphy.

Seitz, R. **Temporal arteritis as a cause for sudden blindness.** Klin. Monatsbl. f. Augenh. **132**: 383-403, 1958.

Seven patients are described of whom six showed a papilledema during the acute stage. All of them had a high sedimentation rate. Treatment with cortisone, antibiotics and vasodilators improved vision in three patients. The improved vascularization was evaluated by ophthalmodynamometry. (11 figures, 2 tables, 47 references) Frederick C. Blodi.

Stankovic, J. and Plavsic, C. **Dissimilar effects of ganglionic blocking agents on the retinal arterial pressure as compared to the blood pressure in hypertensive persons.** Bull. mém. Soc. franç. d'opht. **70**: 346-356, May, 1957.

A series of hypertensive patients were given 100 mg. pendiomide and were kept in a recumbant position during the tests. For two hours the retinal and humoral blood pressure was taken every 10 minutes. The pulse was checked simultaneously. The ocular tension was taken at the beginning of the experiment, one hour later and at the end of it. The patients had either a compensated or a decompensated hypertension. No parallelism could be found in the pressures in the retinal arteries and in the general arterial network after ganglionic blocking, the cerebral vascular system being less dependant on

central sympathetic stimulation. This discrepancy between general and cerebral circulation has been observed during numerous physiologic experiments. (5 figures, 20 references) Alice R. Deutsch.

19

CONGENITAL DEFORMITIES, HEREDITY

Centerwall, W. R. and Miller, M. M. **Ataxia, telangiectasia, and sinopulmonary infections.** A.M.A. J. Dis. Child. **95**:385-396, April, 1958.

A syndrome of slowly progressive deterioration in childhood consisting of progressive ataxia, progressive symmetrical exposure (sun) telangiectasia of the skin and sclera, nystagmus, and chronic recurrent infections of the lung, is presented in three cases. It is probably a heredofamilial disorder transmitted by means of a simple autosomal recessive gene. (28 figures, 21 references) Irwin E. Gaynon.

Delmarcelle, J. and Pivont, A. **Staphyloma corneae, of dominant hereditary transmission.** Bull. Soc. belge d'opht. **117**: 560-568, 1957.

The congenital staphyloma of the cornea is a serious malformation of the anterior segment of the eye of undetermined etiology. By some authors it is ascribed to severe intra-uterine inflammations during the first trimester of gestation. Others believe that the abnormality is caused by an aberration of the mosoderm and is possibly hereditary. The observations of the author concern a five-months-old girl with bilateral congenital staphyloma. Her grandmother and four of her brothers and sisters probably had a unilateral cryptophthalmos. A first cousin had a unilateral corneal staphyloma. The eyes of the little girl had to be removed. The pathologic findings confirmed the absence of mesodermal structures, of Descemet's membrane, endothelium, and iris stroma. The lens was also missing, indicating either a failure of formation or early involution of the lens vesicle. There were no signs of past inflammation. (4 figures, 12 references) Alice R. Deutsch.

Echeverria, R. and Vidal, S. **Tay-Sachs disease.** Arch. chil. de oftal. **39**:106-109, July-Dec., 1957.

The authors review briefly the different forms of this disease, the infantile form, the late infantile and the juvenile form. They then describe in detail the case history of a patient with typical Tay Sachs disease, in whom the diagnosis was correctly made on the basis of the ophthalmoscopic findings, which are very characteristic. (6 references) Walter Mayer.

François, J. **A new syndrome. Dyscephaly, bird-head, dental anomalies, nanism, hypertrichosis, skin atrophy, microphthalmos and congenital cataract.** Bull. Soc. belge d'opht. **117**:569-597, 1957.

The syndrome discussed in this paper is one of the many ectodermal or mesodermal dysplasias ascribed to severe intrauterine disturbances during the 5th, 6th, and 7th week of pregnancy. It is a well defined symptom-complex of dyscephaly, dyscrania, mandibular aplasia, dental anomalies, dwarfism, hypotrichosis, atrophy of the skin, microphthalmos, and congenital cataract. There is apparently no hereditary tendency. The sydrome must be differentiated from progeria, and the essential dyscephalies, cleidocranial dysostosis, dysostosis mandibulo-facialis and dysostosis oto-mandibularis.

Eleven cases gathered from the literature are cited and one personal observation of a 13-year-old boy is reported in detail. (5 figures, 3 tables, 33 references) Alice R. Deutsch.

Howard, Jed Lee. **Mandibulofacial dysostosis (Franceschetti sydrome).** A.M.A. Arch. Ophth. **59**:882-884, June, 1958.

Howard reviews the literature and re-

ports a case. (3 figures, 13 references)
G. S. Tyner.

Luc, Jan. **Sturge-Weber's syndrome. Report of an unusual case.** Brit. J. Ophth. **42**:296-305, May, 1958.

The Sturge-Weber syndrome is characterized by three main features: vascular nevus, mostly in the trigeminal area of the face, radiologic evidence of cerebral calcifications, and convulsions. It is suggested that the condition originates in early fetal life many weeks before the development of the trigeminal nerve and therefore the distribution of the nevus must be accidental but this sign is almost universally present. Glaucoma of one or both eyes is very frequently present. A close relationship seems to exist between this disease and the diseases of von Recklinghausen, von Hippel-Lindau and Bourneville and several others which may actually be abortive forms of this same disease.

A 24-year-old man is described who complained of progressive diminution of vision and who had a generalized nevus over the face and trunk. His pupils were pear-shaped and he had marked glaucoma in one eye and severe papilledema in the other. Intracranial examination revealed a large melanoma which was removed but which later developed metastases. The patient died and was considered to have had Sturge-Weber Syndrome. (4 figures, 33 references) Morris Kaplan.

Meucci, M. and Giordano, R. **A case of congenital bilateral absence of ocular abduction with facial diplegia (Moebius syndrome).** Riv. oto-neuro-oftal. **32**:634-642, Nov.-Dec., 1957.

The authors point out that congenital facial diplegia is a very rare syndrome and that its interpretation is not a simple matter. The authors' patient was a 19-year-old girl who had had bilateral symmetrical paralysis of the muscles innervated by the facial nerves since birth. Ocular examination revealed bilateral paralysis of the lateral rectus muscles.
William C. Caccamise.

Nayrac, P., Graux, P., François, P. and Rabache, R. **Friedreich's disease with optic atrophy and imbecility affecting boys only in a family.** Rev. Neurol. **97**: 295-307, Oct., 1957.

In this family of nine living children the six girls are normal except for esotropia in one, and the three boys show serious degenerative neurologic disease. A rather detailed genealogy is presented and a sprinkling of less grave defects is seen, such as absent tendon reflexes, club foot, scoliosis, strabismus, and mental retardation. The mode of inheritance in the several related and overlapping types of heredofamilial ataxias and their "formes frustes" are discussed. In this particular family the authors prefer to describe the inheritance as recessive with sex-predominance, rather than as recessive sex-linked. Some way must be found to prevent marriages between individuals from such families. (6 figures, 39 references)
Edward U. Murphy.

20

HYGIENE, SOCIOLOGY, EDUCATION, AND HISTORY

Snyder, Charles. **A bibliography of the history of ophthalmology. 1955 and 1956.** A.M.A. Arch. Ophth. **59**:885-894, June, 1958.

A valuable bibliography has been prepared by the librarian of the Lucien Howe Library of Ophthalmology.
G. S. Tyner.

NEWS ITEMS

Edited by DONALD J. LYLE, M.D.
411 Oak Street, Cincinnati 19, Ohio

News items should reach the editor by the 10th of the month. For adequate publicity, notices of postgraduate courses and meetings should be received three months in advance.

DEATHS

Dr. Theodore Felix Paprocki, Ogdensburg, New York, died April 30, 1958, aged 47 years.

ANNOUNCEMENTS

NEW YORK COURSES

New York University Post-Graduate Medical School offers the following courses this fall:

OPHTHALMIC PLASTIC SURGERY, a part-time course of five days' duration, 2:00 to 5:00 P.M., November 17 through 21, 1958, under the direction of Dr. Sidney A. Fox. This course covers the essentials of ophthalmic plastic surgery for the practicing ophthalmologist. Instruction is by lecture and demonstration. Tuition: $85.00.

This course will be repeated 9:00 A.M. to 12:00 M., March 16 through 20, 1959.

SURGERY OF THE CORNEA, a full-time course of five days' duration, December 1 through 5, 1958, under the direction of Dr. Ramon Castroviejo. This course is designed to cover current concepts and practices in the field of corneal surgery. Afternoons from 2:00 to 5:00 P.M. are devoted to didactic lectures, illustrated by lantern slides and motion pictures. During the mornings, as patient material is available, opportunity is offered to observe surgical procedures in the operating room. On the last morning of the course, students perform surgical procedures on animal eyes. Tuition: $225.00.

EASTERN SECTION MEETING

The Eastern Section of the Association for Research in Ophthalmology will meet November 21st and 22nd at Bellevue Hospital, New York. Abstracts of papers proposed for presentation should be sent to Dr. Jerry Hart Jacobson of the program committee before September 15, 1958.

NEW YORK ALUMNI

The annual spring meeting of the Alumni Association of the New York Eye and Ear Infirmary in April, 1958, was so well received that it has been decided to expand next year's meeting which will take place from April 20 to 23, 1959.

Symposia and lectures on pathology, traumatic surgery, and medicolegal ophthalmology will be conducted. It is also planned to offer refresher courses in gonioscopy and tonography, electrophysiology, radio-isotopes, orthoptics, macular function testing, and biomicroscopy. Closed-circuit TV of surgical operations will also be shown.

More complete information regarding the meeting will appear in a later issue of THE JOURNAL.

MISCELLANEOUS

THE GONIN FESTIVAL

The "Gonin Festival" was held at the Palais de Rumine in the senate chamber of the University of Lausanne on July 12, 1958.

The procedure for the allocation and presentation of the Gonin Medal has now become stabilized and this was the first "routine" occasion; it is hoped that it will become an important and pleasant ophthalmologic tradition.

The Gonin Medal in gold is presented every four years conjointly by the University of Lausanne and the Swiss Ophthalmological Society, in memory of their most distinguished ophthalmologic alumnus and member. The medal is given to the person who is internationally considered to have contributed most lavishly to ophthalmology. In order to ensure that the choice is widely acceptable throughout the world, the International Council of Ophthalmology appoints every eight years a commission of seven distinguished ophthalmologists from seven different countries to suggest names of potential candidates; from these the recipient is chosen by the council itself at its meeting prior to each International Congress of Ophthalmology. The medallist visits the University of Lausanne a short time before each congress, delivers an oration, and receives a certificate from the rector of the university entitling him to claim the medal. The medal, itself, is presented at each congress.

On July 12th a very delightful ceremony took place wherein Dr. Edmond Grin, the rector of the university, opened the proceedings. The president of the International Council (Duke-Elder) introduced the Medallist, Prof. Alan C. Woods of The Johns Hopkins University, Baltimore, explaining the reasons for the choice of the council. The professor of ophthalmology of the University of Lausanne (Streiff) detailed the brilliant career of the medallist and his immense contributions to ophthalmology. Thereafter Professor Woods delivered the Gonin Oration.

CENTENNIAL DINNER FOR ILLINOIS EYE AND EAR INFIRMARY

The Illinois Department of Public Welfare, in co-operation with the University of Illinois and the ophthalmologic and otolaryngologic alumni associations, will hold a centennial celebration dinner for the Illinois Eye and Ear Infirmary in the Grand Ballroom, Palmer House, Chicago, at 7:00 P.M. (DST) on Wednesday, October 15, 1958.

Guest speaker for the dinner will be Vice Ad-

miral Ross T. McIntire, surgeon general of the United States Navy (retired), whose subject will be "Medicine in our changing times."

Dr. Otto L. Bettag, director, Illinois Department of Public Welfare, will serve as toastmaster for the occasion.

According to Lester R. Gerber, superintendent of the Illinois Eye and Ear Infirmary and chairman of the centennial celebration committee, an attendance of more than 750 persons is expected, including members of the American Academy of Ophthalmology and Otolaryngology who will be holding their annual meeting in Chicago during the week beginning October 13th.

Persons interested in attending the centennial celebration dinner may write Mr. Gerber at the Infirmary for tickets. Address: Illinois Eye and Ear Infirmary, 904 West Adams Street, Chicago 7, Illinois.

Societies

Colorado Ophthalmological Society

New officers of the Colorado Ophthalmological Society for 1958-1959 elected at the annual business meeting are: President, Dr. James C. Strong, Jr.; vice-president, Dr. Morris Kaplan; secretary, Dr. Max Kaplan; treasurer, Dr. George A. Filmer; recording secretary, Dr. Mitchell B. Rider; member of the executive committee, Dr. Daniel Franklin.

Section prizes

The annual scientific session of the Section on Ophthalmology, American Medical Association, was held in San Francisco from June 24 to 26, 1958. Approximately 400 persons, a record for attendance on the West Coast, were present throughout the meeting. Prof. Gösta Karpe of Stockholm, Sweden, was the guest-of-honor of the section.

The following prizes and honors were awarded:

1. The Prize Medal in Ophthalmology was awarded to Peter C. Kronfeld for his many contributions to our specialty.

2. The $250 prize for the best exhibit was awarded to L. K. Garron, M. J. Hogan, W. K. McEwen, and M. L. Feeney for their exhibit, "Electron microscopy of ocular tissue."

3. The $250 prize was awarded to Warren A. Wilson for his paper, "Galactosemia with associated cataracts in children," judged on the basis of presentation and originality.

The following officers were elected: Frank B. Walsh, chairman; Philip M. Lewis, vice-chairman; Henry F. Allen, assistant secretary; Ralph O. Rychener, delegate to House of Delegates; W. Howard Morrison, alternate delegate to House of Delegates; Frank W. Newell, representative to scientific exhibit.

Denver meeting

At the summer Convention of the Colorado Ophthalmological Society held recently in Denver in conjunction with the Office of Postgraduate Medical Education of the University of Colorado School of Medicine, a symposium on cataract was presented. Ophthalmologists participating were Dr. Jerome W. Bettman, San Francisco; Dr. Jack S. Guyton, Detroit; Dr. Frederick R. Carriker, Phoenix; Dr. Jose A. Quiroz, Mexico City; and Dr. John C. Long, Denver.

THE CHICAGO OPHTHALMOLOGICAL SOCIETY
1959 CLINICAL CONFERENCE

February 13 and 14, 1959—The Drake Hotel, Chicago, Illinois

SPEAKERS:

Paul A. Chandler, M.D.	J. Robert Fitzgerald, M.D.	Charles Oldfield, Jr., M.D.
Charles E. Iliff, M.D.	William F. Hughes, M.D.	John H. Olwin, M.D.
Samuel J. Kimura, M.D.	Gilbert Iser, M.D.	Frank Pirrucello, M.D.
John Harry King, M.D.	Bertha Klien, M.D.	Theodore Zekman, M.D.
J. Vernal Cassady, M.D.	Frank W. Newell, M.D.	

SUBJECTS:

Symposium: Management of Surgical Conditions of Lids and Lacrimal Apparatus.
Management of Uveitis • Plastic Surgical Principles
Prophylaxis and Treatment of Vascular Occlusions • Clinical Value of Electroretinography
Management of Cardiac Arrest • Glaucoma Management
Present Status of Preserved Eye Tissue for Transplantation.

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THE FIFTEENTH ANNUAL GIFFORD MEMORIAL LECTURE

Paul A. Chandler, Boston, Massachusetts • Friday, February 13, 1959, The Drake Hotel • 5:15 P.M.

The Chicago Ophthalmological Society welcomes all physicians to attend the Lecture. A buffet supper which follows is open to physicians and their wives.

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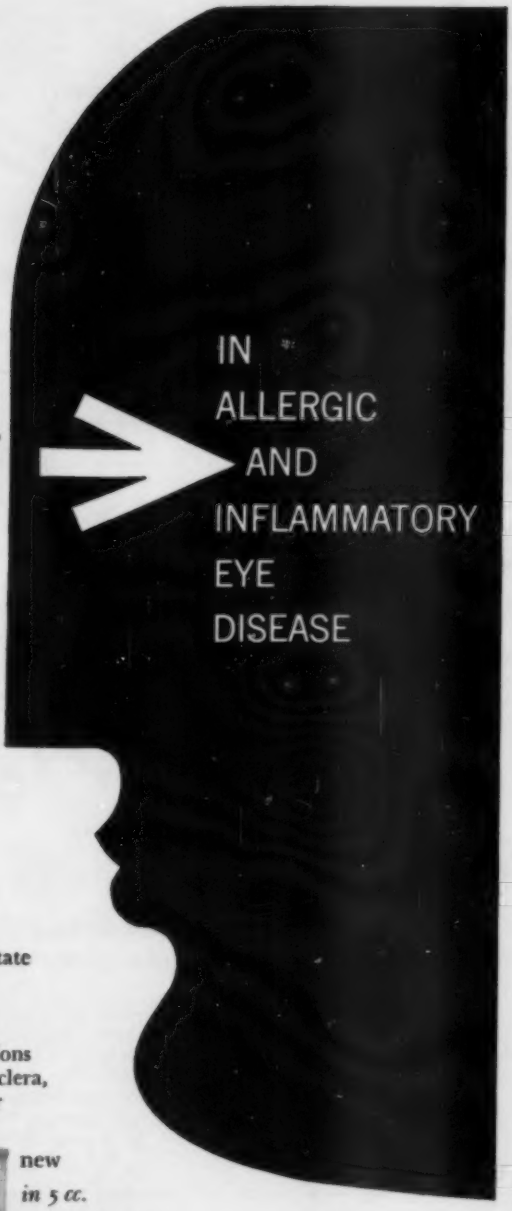

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